RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

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RADIOLOGY

A MONTHLY PUBLICATION DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

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PUBLISHED BY THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

Vol. 53 NOVEMBER 1949 No. 5

Roentgen Demonstration of Displaced Intracranial Physiologic Calcification and Its Significance in the Diagnosis of Brain Tumors and Other Space-Occupying Diseases[1]

BARTON R. YOUNG, M.D.

Philadelphia, Penna.

THE PURPOSE OF this paper is to re-emphasize the significance of displaced physiologic intracranial calcification in the diagnosis of space-occupying disease and thus stimulate greater interest in the demonstration and recognition of normally and abnormally located calcifications by roentgen study.

Ever since the pineal was demonstrated by roentgen methods (Fig. 1A), its importance as an intracranial landmark has been appreciated. More than three decades ago Schüller (1) stated that a diagnosis of space-taking disease was indicated when roentgenograms revealed pineal displacement.

In 1925, in order to determine in just what percentage of persons the pineal is calcified, Naffziger (2) studied roentgenograms of the skull obtained in 215 consecutive cases. He found roentgen evidence of calcification in 45 per cent and therefore advocated stereoscopic studies, especially in the anteroposterior projection, in the presence of increased intracranial pressure, as a means of ascertaining the position of the gland. He reported lateral displacement of the pineal in 2 patients with brain tumors. Excellent articles by Vastine and Kinney (3) and later by Dyke (4) and Fray (5) dealing with pineal position as determined by the lateral view, stimulated the adoption of a routine determination of pineal position by many radiologists.

Vastine and Kinney established the normal pineal zone in relation to the anterior and posterior extremities of the cranium by measuring, on the lateral films of several hundred normal persons, the distance from the inner table of the frontal bone to the pineal and plotting this measurement against the anteroposterior dimension of the skull. The normal pineal zone in relation to the vertex and base was determined by plotting the distance from the inner table of the vault to the pineal against the vertex-base dimension (Fig. 1, B and C).

By use of the Vastine-Kinney measurements and charts, it is easy to determine whether the pineal is in normal relation to the extremities of the cranium as measured on the lateral film. When the pineal is outside its normal zone, a space-occupying lesion is the usual cause. The approximate position of the lesion is determined by the direction of displacement of the pineal.

Dyke (4) determined the position of the pineal in 3,000 consecutive skull examina-

[1] From the Departments of Radiology, Temple University and Germantown Hospitals, Philadelphia, Penna. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

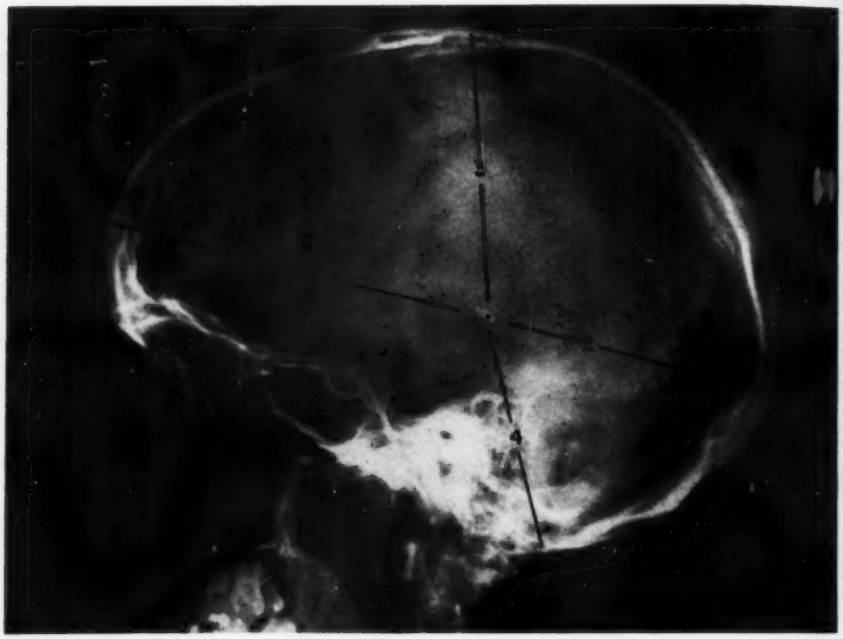
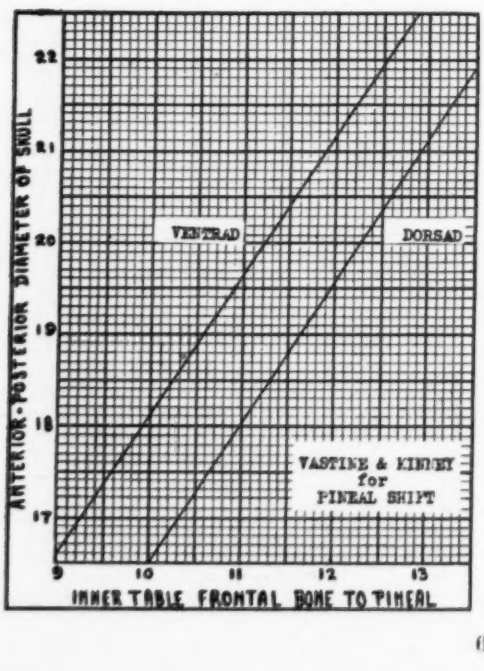


Fig. 1A. Vastine-Kinney measurements. The distance (measured in cm.) from the inner table of the frontal bone to the pineal (1) is plotted against the anteroposterior dimension of the skull (1 plus 2) and the measurement from the inner table of the vault to the pineal (3) is plotted against the vertex-base dimension (3 plus 4). When the pineal is spotted between the oblique lines in the charts (Fig. 1, B and C) it is in the normal zones.



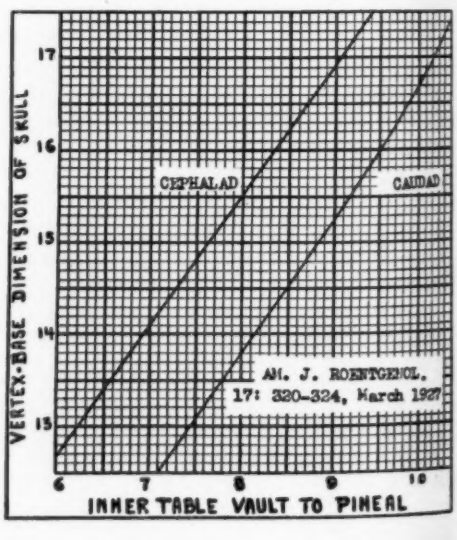


Fig. 1, B and C. Vastine-Kinney charts. When the pineal is spotted between the oblique lines, it is in the normal zones.

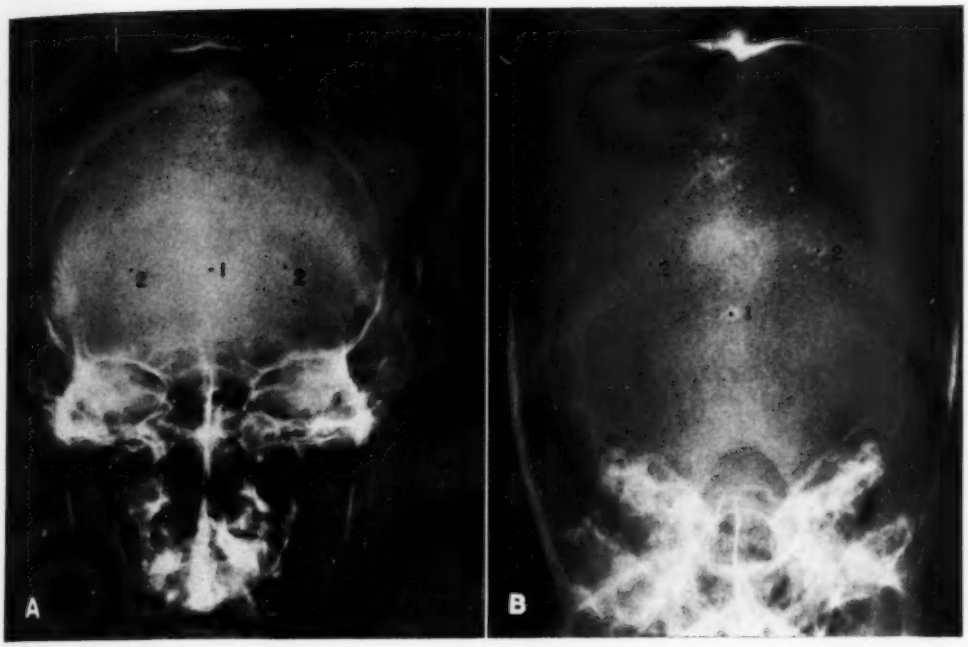


Fig. 2. Normal pineal and choroid plexus calcifications. The relation of the midline pineal calcification (1) to the choroids (2) is demonstrated in the frontal (A) and occipital views (B). In the occipital view the choroids (B, 2) are projected above the level of the pineal (1).

tions, using this method, and after carefully correlating the results with the clinical and other findings, concluded that they were reliable. He found the pineal slightly in front of the Vastine-Kinney zone in a number of normals, but otherwise his results were the same.

A few years later Fray (5) advocated different methods of measurement on the lateral film which clearly indicate whether the pineal is in normal relation to the extremities of the cranium. His method yields good results and is preferred by some.

Further information about the position of the pineal is obtained when it is demonstrated by either frontal or occipital projections, because its relation to the sides of the cranium is revealed and space-occupying disease is thus lateralized (Fig. 2, A and B, 1). Stereoscopic views are often necessary in order to determine whether the visible calcific shadow is actually in the pineal. They are indicated when the latter

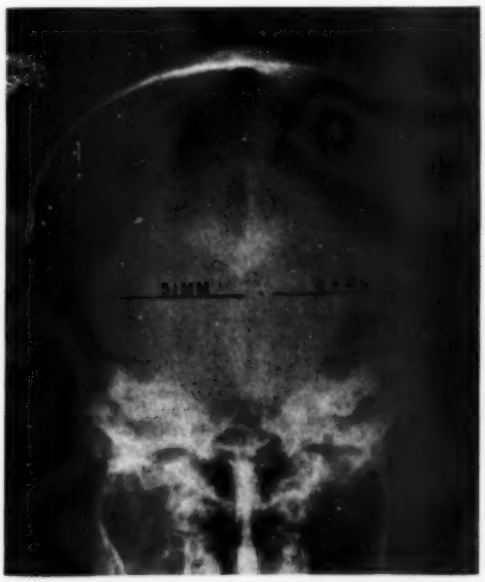


Fig. 3. Pineal displacement by metastatic bronchial carcinoma. The tumor displaced the pineal 3.5 mm. to the opposite side. Top normal displacement is 2.5 mm. from the midline.

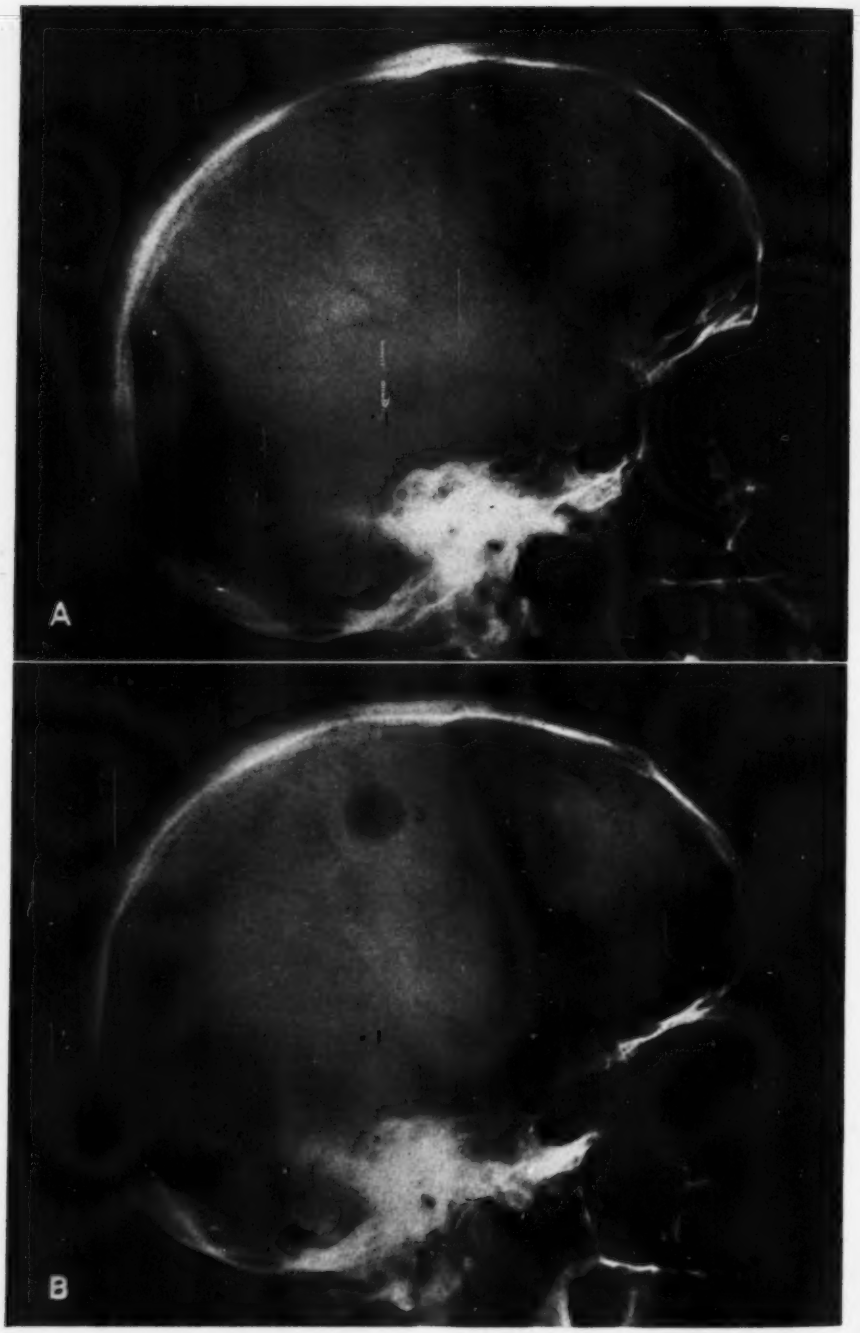


Fig. 4. Downward and posterior displacement of the pineal by a large subdural hematoma covering both cerebral hemispheres (A, 1). The pineal returned to its normal position (B, 1) after the clot was evacuated through trephine openings (B, 3). Pressure decalcification of the dorsum sellae (A, 2; B, 2) disappeared several months after surgery.

is not demonstrable in non-stereoscopic views.

Normally the pineal is situated midway between the sides of the cranium (Fig. 2). Occasionally it is found as far as 2 mm., and rarely 2.5 mm., from the mid-sagittal plane of the cranium in the absence of clinical evidence of intracranial disease. A deviation of more than 2.5 mm. is indicative of significant abnormality, usually a space-taking lesion (Fig. 3).

Determination of the position of the pineal is important when the clinical findings are borderline and there is no evidence of bone erosion and abnormal calcification to suggest intracranial disease. Abnormal pineal position necessitates further investigation, while normal findings are reassuring.

It is of even greater importance to determine the location of the pineal when the neurologic findings indicate an expanding lesion and contrast studies are considered hazardous because of the critical condition of the patient.

Pineal displacement is most frequently on the basis of a primary brain tumor. Glioma outranks all other primary tumors as a cause of pineal displacement, with meningioma second in incidence (4). Not infrequently a metastatic tumor is responsible. For this reason, every patient with evidence of a brain tumor should have a complete investigation of all systems, including roentgen studies to exclude a primary lesion elsewhere before craniotomy is done (Fig. 3). Since 65 per cent of metastatic brain tumors arise in the lung and breast, these areas should be investigated primarily.

Next in order after tumor as a cause of pineal displacement is a hematoma, usually subdural in location. Subdural hematoma is nearly always traumatic. Frequently evidence substantiating it is lacking unless the hemorrhage is large enough to displace the pineal or is calcified. In the patient whose films are reproduced in Figure 4, a clinical diagnosis of subdural hematoma was supported by finding the pineal displaced downward and posteriorly (A, 1). After evacuation of the clot, the pineal returned to its normal position (B, 1).

Fig. 5. Brain abscess displacing the pineal 7 mm. from the midline. Clinical diagnosis of temporal lobe abscess of otitic origin. Air was injected into cavity after drainage (1).

Intracerebral hematomas often attain sufficient size to displace the pineal, and this condition must therefore be considered in the differential diagnosis. In two of three temporal lobe hematomas, Scott (6) found the pineal displaced significantly from the midline to the opposite side of the brain. Less frequently found space-taking lesions that may cause pineal displacement are subdural hygroma, brain abscess (Fig. 5), cyst, and granuloma.

The choroid plexuses of the lateral ventricles are relatively poor landmarks in comparison to the pineal, since the incidence of calcification in these structures, before the age of fifty, is low, and it is frequently asymmetrical and unilateral. Since calcification normally occurs at different levels in the choroids, it is difficult to be certain whether mild variations in their position are significant. However,

Fig. 6. Downward and forward displacement of choroid calcification (1) by a tumor. The choroid in the other lateral ventricle (2) is slightly displaced in opposite directions.

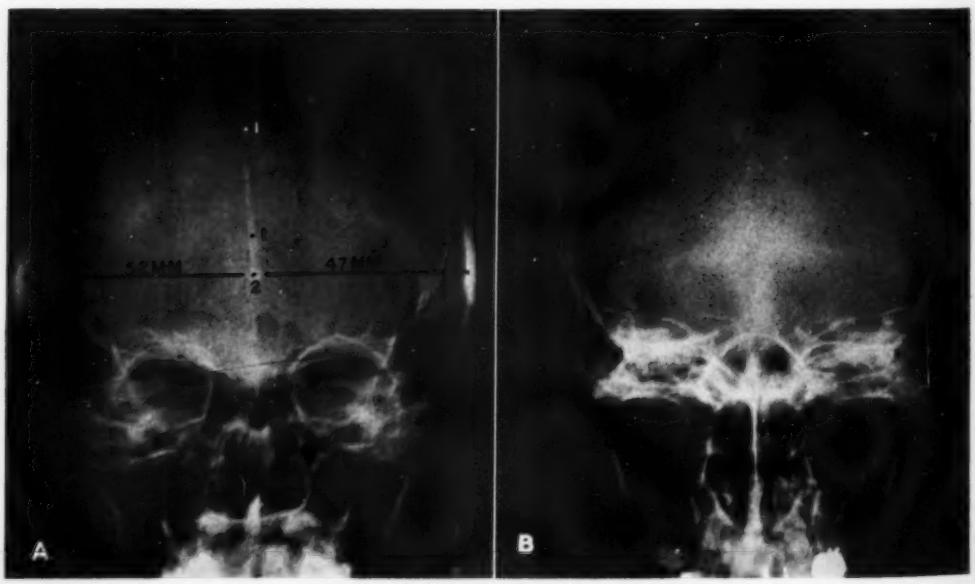


Fig. 7. Falx and pineal displacement (A, 1 and 2) by a tumor. Compare the sharp borders of physiologic calcification (A, 1) with the irregular margins of calcification in a meningioma (B, 1).

a marked change in position of a well calcified choroid must be considered suggestive of tumor and evaluated in relation to the clinical and other roentgen findings (Fig. 6).

When a calcified choroid is displaced sufficiently to indicate space-taking disease, other roentgen findings, as sellar erosion and pineal displacement, are often evident. This is not always true, however, as emphasized in an excellent article by Childe (7). He reports that in 3 of 8 patients with displacement of a calcified choroid by an expanding lesion, the diagnosis was based on this finding alone, since the pineal was not calcified nor the sella eroded.

The skull roentgenograms of two cases with expanding intracranial lesions reported by Wood (8) showed no other abnormality than displacement of a calcified choroid. His review of the literature revealed that most of the lesions which caused such displacement were posterior to the choroid or in the temporal lobe. In one case, previously reported (9), a calcified choroid was displaced downward and forward by a large tumor (Fig. 6).

Physiologic calcification in the falx is infrequently helpful in the diagnosis of space-occupying disease, because it is usually manifest as a few small plaques near the vertex. Displacement of extensive sheath-like calcification in the falx by an expanding lesion is uncommon but, when present, is well demonstrated by frontal (Fig. 7A, 1) and occipital views.

It is important to differentiate physiologic calcification in the dura from calcification on the basis of meningioma, especially in the vertex and falx regions. Physiologic calcification is linear and its margins are usually sharp in contrast to the fuzzy borders of calcification due to meningioma (Fig. 7B, 1).

SUMMARY AND CONCLUSIONS

The roentgen demonstration of physiologic calcification is an important diagnostic aid in the recognition and evaluation of space-taking disease in the cranium. Calcification occurs in the pineal more frequently than in other intracranial structures and therefore more information is obtained by a study of its position than of the choroids and falx. Every effort should be made to demonstrate calcification in the pineal, not only in lateral views but also in frontal and occipital views. For the latter two projections, stereoscopic studies are often essential, and such studies are more informative than single views for all projections.

Displacement of the pineal from the midline of more than 2.5 mm. indicates significant intracranial abnormality, usually space-occupying disease. Displacement of the pineal, choroids of the lateral ventricles, and falx by various space-taking diseases is demonstrated and discussed.

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REFERENCES

1. SCHÜLLER, A.: Roentgen Diagnosis of Diseases of the Head. Eng. trans. by F. F. Stocking. St. Louis, C. V. Mosby Co., 1918.
2. NAFFZIGER, H. C.: Method for Localization of Brain Tumors—The Pineal Shift. Surg., Gynec. & Obst. **40:** 481–484, April 1925.
3. VASTINE, J. H., and KINNEY, K. K.: Pineal Shadow as Aid in Localization of Brain Tumors. Am. J. Roentgenol. **17:** 320–324, March 1927.
4. DYKE, C. G.: Indirect Signs of Brain Tumor as Noted in Routine Roentgen Examinations; Displacement of Pineal Shadow. A Survey of 3000 Consecutive Skull Examinations. Am. J. Roentgenol. **23:** 598–606, June 1930.
5. FRAY, W. W.: A Roentgenological Study of Pineal Orientation III. A Comparison of Methods Used in Pineal Orientation. Am. J. Roentgenol. **39:** 899–907, June 1938.
6. SCOTT, M.: Surgical Treatment of Spontaneous Nontraumatic Hematomas. J. A. M. A. **130:** 845–850, March 30, 1946.
7. CHILDE, A. E.: Calcification of the Choroid Plexus and Its Displacement by Expanding Intracranial Lesions. Am. J. Roentgenol. **45:** 523–536, April 1941.
8. WOOD, E. H., JR.: Some Roentgenological and Pathological Aspects of Calcification of the Choroid Plexus. Am. J. Roentgenol. **52:** 388–398, October 1944.
9. YOUNG, B. R.: The Skull, Sinuses and Mastoids. A Handbook of Roentgen Diagnosis. Chicago, The Year Book Publishers, Inc., 1948.

(For Spanish summary, see following page)

SUMARIO

Demostración Roentgenológica de la Calcificación Fisiológica Intracraneal Desplazada e Importancia de la Misma en el Diagnóstico de los Tumores y Otras Afecciones Emplazadas del Cerebro

El descubrimiento roentgenológico de la calcificación fisiológica constituye un importante auxiliar diagnóstico en el diagnóstico y justipreciación de toda enfermedad que ocupa espacio en el cráneo. La calcificación es más frecuente en la glándula pineal que en otros tejidos intracraneales, por lo cual se obtiene más información estudiando la posición de la misma que la de los plexos coroideos y la hoz. Debe hacerse todo lo posible para descubrir la calcificación pineal, no sólo en las vistas laterales, sino también en las frontales y occipitales. Para estas dos últimas proyecciones, los estudios estereoscópicos resultan a menudo indispensables, siendo más informativos que las vistas aisladas en todas las proyecciones.

Un desplazamiento de la pineal de más de 2.5 mm. de la línea media indica una importante anomalía intracraneana, por lo general enfermedad que abarca espacio, y más especialmente glioma, meningioma o hematoma. El desplazamiento de los plexos coroideos de los ventrículos laterales, cuando se descubre, indica igualmente lesión que ocupa espacio. La calcificación de la hoz es menos útil en el diagnóstico por soler manifestarse únicamente en forma de algunas plaquillas cerca del vértice.

Eighth Nerve Tumors: Their Roentgen Manifestations[1]

PHILIP J. HODES, M.D., EUGENE P. PENDERGRASS, M.D., and BARTON R. YOUNG, M.D.
Philadelphia, Penna.

EIGHTH NERVE tumors account for about 6 per cent of all intracranial neoplasms (12, 18). As they are particularly suited to surgical removal, their early recognition is essential. The present study was undertaken to see how often we, as radiologists, help in their diagnosis.

Fig. 1. Normal and symmetrical internal auditory canals seen in the postero-anterior projection.

The data upon which this report is based were taken from the records of the Neurosurgical Services of the Hospital of the University of Pennsylvania, the Graduate Hospital of the University of Pennsylvania, and the Temple University Hospital, made available to us through the kindness of the neurosurgeons of these hospitals, Dr. Francis C. Grant, Dr. Robert A. Groff, and Dr. Michael Scott.

Records of 129 patients with proved 8th nerve tumors were reviewed. Of these, 83 were operated upon at the Hospital of the University of Pennsylvania, 29 at the Graduate Hospital of the University of Pennsylvania, and 17 at the Temple University Hospital. In 122 cases the verbal descriptions of the roentgen findings were considered satisfactory for analysis, and in 70 we personally reviewed and studied the roentgenograms.

The purposes of the present communication are, first, to review the literature concerning the anatomy, pathology, and clinical findings in 8th nerve tumors, and second, to record their roentgen manifestations.

ANATOMY

The 8th nerve enters the brain at the inferior border of the pons. It contains

[1] From the Departments of Radiology of the Hospital of the University of Pennsylvania and the Temple University Hospital. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

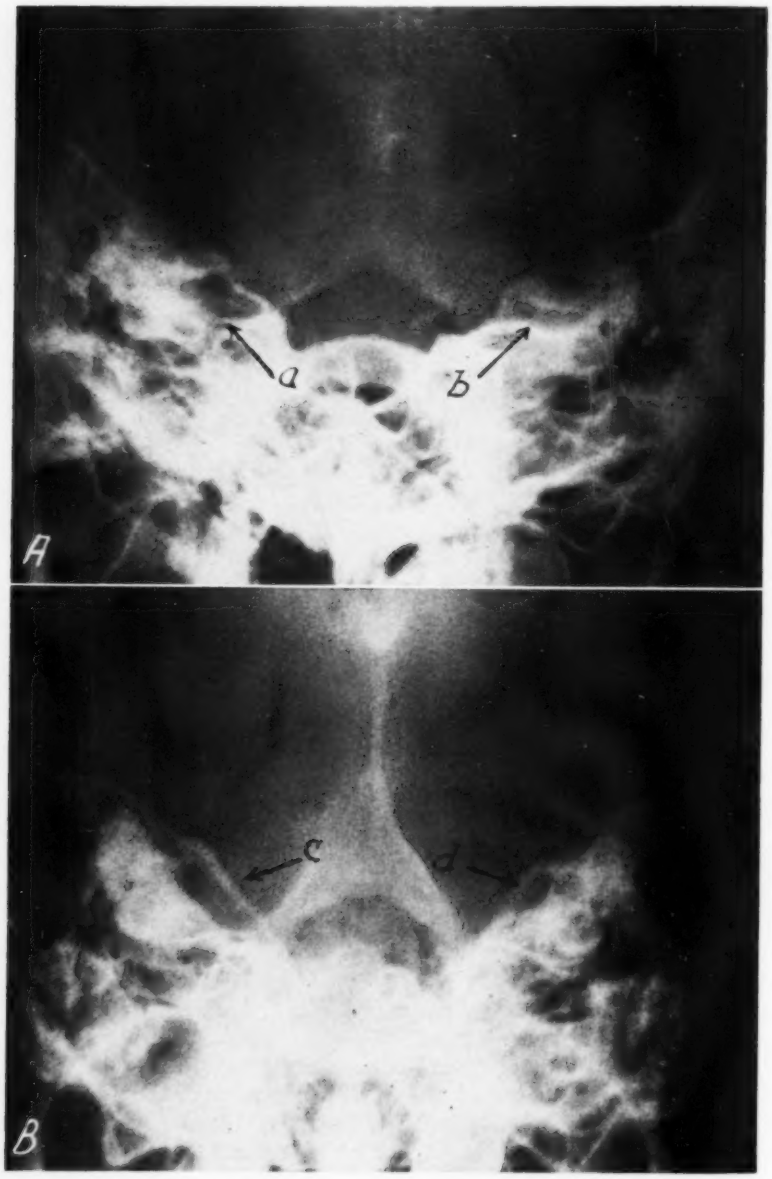

Fig. 2. Normal and asymmetrical internal auditory canals seen in the occipital projection.
A. The right internal auditory canal, *a*, is wider than *b*.
B. The right internal auditory canal, *c*, is wider than *d*.

two groups of fibers, those supplying the cochlea, concerned with hearing, and those which supply the semicircular canals, the utricle, and saccule, which are concerned with posture and equilibrium. These portions of the acoustic nerve are known, respectively, as the cochlear nerve and the vestibular nerve (12, 11, 3).

As the nerve passes from the internal auditory canal to the brain stem, it courses

Fig. 3. Normal and asymmetrical internal auditory canals seen in the postero-anterior projection.
A. The right internal auditory canal, *a*, is wider than *b*.
B. The right internal auditory canal, *c*, is wider than *d*.

through a space below the tentorium in the posterior fossa known as the "cerebellopontile angle." Called more specifically the "subtentorial angle" by Stibbe (34), it is bounded above and laterally by the sloping rigid tentorium, below and laterally by the sloping posterior surface of the petrosa, and above and medially by the inferior surface of the cerebellum and the side of the pons. This portion of the sub-

arachnoid space measures about 1/2 inch in width and 1 inch in depth. It contains cerebrospinal fluid in a fine network of trabeculae which stretch from the arachnoid on the bone to the pia mater on the cerebellum and pons. Crossing this space are the 5th, 7th, and 8th nerves, the nervus intermedius, and the anterior inferior cerebellar artery and vein. The cochlear division of the acoustic nerve lies slightly behind the vestibular branch, but the entire bone resorption reflect the tightness of the cerebellopontile angle.

The roentgen anatomy of the petrous pyramid, as well as its many variants, is of obvious concern to men interested in acoustic tumors. They well appreciate how different the petrosae appear in different people, and indeed, in the two sides of the head in the same individual (Figs. 1–4).

This problem was studied extensively by Ebenius in 1934 (14) and more recently by

Fig. 4. Body-section examination of normal and asymmetrical internal auditory canals. The left internal auditory canal, *b*, is wider and shorter than the right, *a*. (*Note:* This patient complained of tinnitus in the left ear and subsequently tinnitus in the right ear. Neurological examinations and Barany tests were negative. The tinnitus was finally considered of allergic origin.)

8th nerve, 7th nerve, and nervus intermedius course as a group through the angle into the internal auditory canal (34).

One need but think of the many structures crowded together into the subtentorial angle to realize why 8th nerve tumors so often affect neighboring cranial nerves. The common occurrence of increased intracranial pressure with compression of the pons and cerebellum attest still further to the constricted nature of the space. Of obvious importance to radiologists are the changes in the internal auditory canal and petrosal apex, where varying amounts of

Camp and Cilley (5). By observations on hundreds of normal heads, these men showed statistically how varied the measurements and configuration of petrosal pyramids could be. Ebenius, reviewing the films of 100 normal heads, found both acoustic meati alike in 41 to 67 per cent, depending upon the projection in which the films were taken. Camp and Cilley, in 250 pairs of petrosae, found both sides alike in 103, or 41 per cent. When the diameters of both pori of the same skull were compared in the same series, Camp and Cilley found variations of from 0.5 to 1.5 mm. in 125

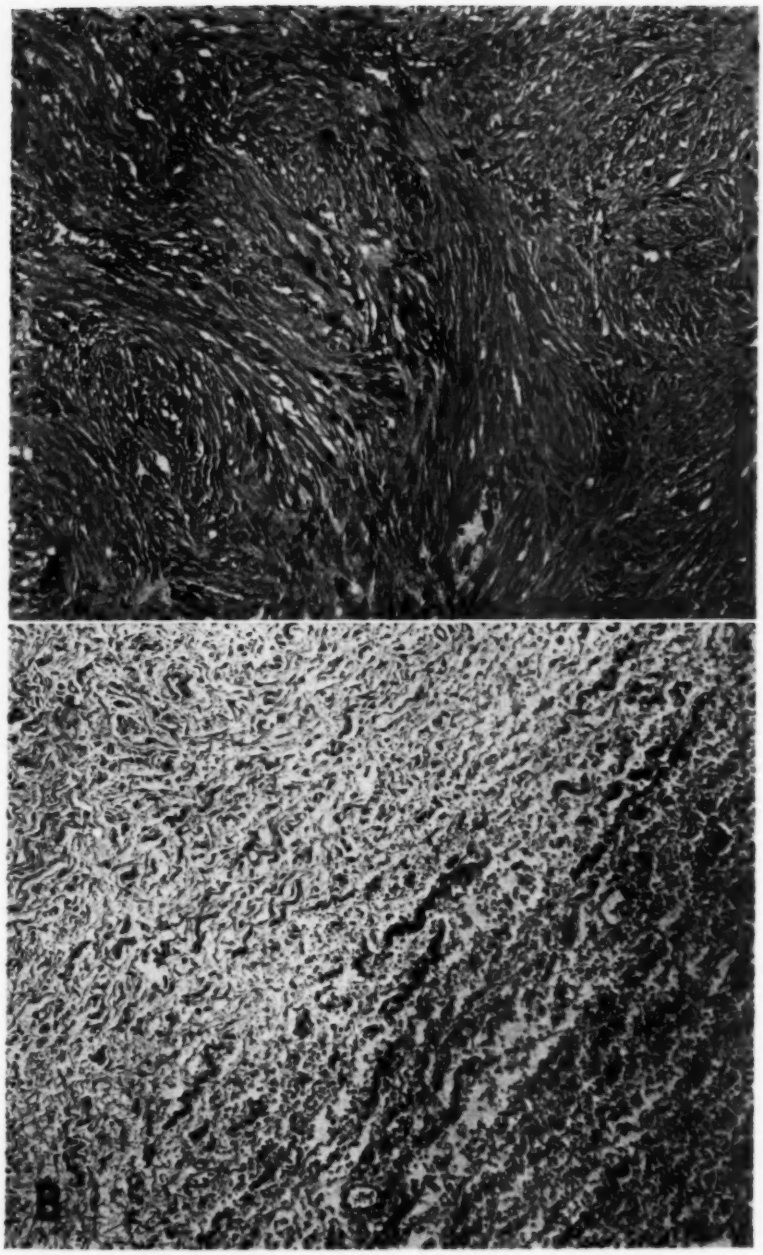

Fig. 5. A. Microscopic section from an acoustic neurinoma, showing the characteristic streaming architecture and palisading of the nuclei.

B. Microscopic section from a neurofibroma, revealing fine amyelinic nerve fibers on the left, a few thick myelinated nerve bundles in the center, and fibromatous element to the right.

Fig. 6. Meningioma in the base of the left frontal lobe, seen at *a*, *b*, and *c*. This was associated with an 8th nerve neurinoma which was removed at operation and is not included in this specimen.

heads. In 22 the variation was from 1.5 to 2.5 mm. (Figs. 2 and 3). Equally marked differences were noted in the shape of the internal auditory canal. In 174 patients, both sides were found to be alike; in 76 they were dissimilar.

Camp and Cilley also determined the depth of the internal auditory canal and found it to average 7.9 mm. on the right side and 7.8 mm. on the left. The deepest canal measured 16 mm., while the shallowest measured 3 mm. The majority were from 4 to 10 mm. in depth. Of the 100 pairs of internal auditory canals compared, the depth was alike in 27. In 38 there was from 1 to 2 mm. difference between the two sides. In 23 pairs the difference in depth was from 2 to 3 mm., and in 7 from 3 to 4 mm. The greatest variation in depth, 5 mm., was noticed only once (Fig. 4).

PATHOLOGY

It is not within the province of this paper to review the histopathology of 8th nerve tumors in detail. Suffice it to say they fall into three large groups. The first, which includes about 90 per cent of the total, consists of the acoustic neurinomas (perineural fibroblastoma). The second group, often familial and bilateral, is made up of von Recklinghausen's neurofibromas. The third group, least common and rather rare, includes von Recklinghausen's disease in association with meningiomas (13).

According to the literature (13, 15, 30), neurinomas and Recklinghausen's neurofibromas are differentiated from each other microscopically by the relationship between the nerve fibers and the tumor capsule. In the neurinomas the preformed nerve fibers are demonstrated only in the capsule of the tumor. In von Recklinghausen's neurofibromatosis the nerve fibers course through and penetrate the tumor (Fig. 5).

Neurinomas potentially can arise from any of the spinal nerve roots. For some reason, when they involve the cranial nerves they almost invariably affect the acoustic nerve. They have also been known to involve the 5th, 10th, and 11th cranial nerves (20).

Henschen (23, 24) early postulated that acoustic neurinomas arise from the vestibular branch of the nerve within the internal auditory canal. He came to this conclusion because of the frequency with which he found complete loss of vestibular responses in patients who were still able to hear, even though they had large acoustic tumors. Henschen's conviction seems to be supported by the important disclosures of Hardy and Crowe (22). Examining serial sections of 250 apparently healthy petrous pyramids, these investigators demonstrated small acoustic tumors in 6. In each instance the acoustic neurinoma lay entirely within the internal auditory canal, apparently intimately connected with its vestibular branch. It seems fair to state that Cushing (12) doubted this as an all-inclusive concept because of the many large acoustic tumors he had seen unattended by changes in the internal auditory canal.

The second group of acoustic tumors constitutes a bizarre form of von Reckling-

hausen's neurofibromatosis (Fig. 23), called "acoustic neurofibroma," rather than "acoustic neurinoma." The lesions are often bilateral and show familial tendencies (12, 13, 20). This familial trait was dramatically illustrated by the Gardner-Frazier family (17–19), in which bilateral deafness was transmitted as a true mendelian dominant to 217 relatives through five generations. As occurs so frequently in the neurofibroma group, the Gardner-Frazier family also revealed multiple cranial nerve involvement, including blindness.

The tumors of the third group, and the least common, also belong to the von Recklinghausen's neurofibroma family, but with them are associated meningiomas, either single or multiple (19). To this group Cushing gave the name "meningiomatosis," as it seemed to suggest more vividly the tendency to dissemination throughout the brain. Attention is called to the fact that meningiomas may be found in patients with acoustic neurinomas as well as neurofibromas, as we observed in one patient and as has been reported by others (12, 19) (Fig. 6).

The fact that leiomyomas may be found also in this group of tumors, suggests that central neurofibromatosis and meningiomatosis may be diseases involving all of the binding tissues of the nervous system and not the nerve sheaths alone (13).

To the radiologist, it probably makes little difference whether an internal auditory canal is being eroded by a neurinoma, neurofibroma, or neurofibroma associated with meningiomas. All apparently affect the petrosa similarly. What is important, however, is that radiologists, cognizant of the pathology of the disease, be alerted to its pleomorphic clinical characteristics, particularly in patients suspected of having bilateral acoustic tumors.

The gross appearance of 8th nerve tumors is rather typical. Lying usually in the subarachnoid space (12), they are somewhat oval in shape and well encapsulated (Figs. 7, 12, 14, 15). They grow slowly and as they become larger tend to conform in shape to the resistant petrosal bone on the one side, to the posterior fossa below, and to the rigid tentorium above (12, 18). On their free side, they crowd and distort the cerebellum and pons. Encysted collections of yellow fluid commonly surmount the growth (33). In rare instances the tumor may do all of its growing within the canal and present as a mass covered by a thin bony wall (1).

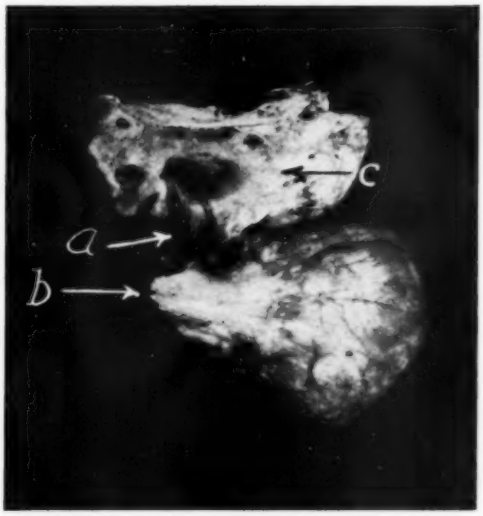

Fig. 7. Postmortem specimen taken from a patient with an 8th nerve tumor. The 8th nerve is seen at *a*, the acoustic tumor at *b*, and the petrosal apex at *c*. The porus acusticus internus is clearly visualized and is considerably dilated. Note the teat-like configuration of the tumor at *b*, where it lay in intimate contact with the porus. (Courtesy Dr. John D. Camp, Mayo Clinic.)

The tumors vary considerably in color and consistency. Usually they are grayish-yellow in appearance. The more fatty degeneration it has undergone, the more yellow is the tumor and the more easily is it enucleated. The grayer tumors are more fibrous and more difficult to remove.

A small teat-like prominence usually exists on the surface of 8th nerve tumors, corresponding in location and configuration to the porus acusticus internus (Figs. 7, 12, 15). According to Cushing (12), this nipple is so characteristic of acoustic tumors that its connection with the meatus could not be mistaken even though the relationship had been overlooked originally.

CLINICAL FINDINGS

Eighth nerve tumors usually follow a characteristic progressive pattern of neurologic signs and symptoms. In most instances, one can consider their pathologic physiology as being divided into three stages (21). In the first stage, the tumor and its symptoms are limited to the 5th, 7th, and 8th nerves. As the tumor grows, the second stage of the syndrome ensues, with ataxia of cerebellar origin. Finally, in the third stage, the clinical manifestations of increased intracranial pressure become evident.

These tumors occur predominantly in middle life (18). It has been the experience of most observers that women are much more commonly affected than men. Of 50 patients reported by Grant and Spitz (21), 33 were women. (All have been included in the present report.)

According to Gardner (18), the average duration of symptoms is two or three years. Grant and Spitz (21) found that the duration varied from about six months to sixteen years.

Since the publication of Cushing's classic monograph on acoustic nerve tumors in 1917, the literature has repeatedly emphasized his chronology of symptoms. Indeed, some consider it almost infallible (28). According to Cushing, the earliest symptoms are auditory and labyrinthine. Tinnitus in the affected ear, followed by gradually progressing deafness, usually ushers in the disease. For a long time the symptoms are confined to the functions of the 8th nerve. As the tumor grows, headache, particularly occipital and occipitofrontal, appears. With pressure upon the cerebellum, pons, and medulla, a mild degree of unsteadiness occurs, gradually assuming the characteristics of cerebellar in-co-ordination and instability. Usually, when the tumor has progressed thus far, evidence of adjacent cranial nerve involvement becomes manifest. As the 5th nerve is affected, patients complain of trigeminal neuralgia or numbness of the face. Facial paresis, paralysis, or even spasm develop as the 7th nerve is included in the growth.

When the tumor has attained sufficient size to interfere with the aqueduct of Sylvius, the signs of increased intracranial pressure ensue. The headaches become worse. Vomiting, ataxia, failing vision, dysarthria, and dysphagia follow soon thereafter. Death commonly is the result of a cerebellar crisis.

Unfortunately, not all patients with 8th nerve tumors present this classical chronology of symptoms. The literature contains accounts of many verified acoustic tumors which do not conform to the rules of the syndrome (16, 20, 25). González (20) found that only 31 per cent of his series followed Cushing's chronology.

Grant and Spitz, reporting 50 8th nerve tumors from the Neurosurgical Service of the Hospital of the University of Pennsylvania, paid particular attention to the first symptom complained of by their patients. In 20 patients, tinnitus plus deafness was the inaugural symptom; in 18 deafness occurred alone. Vertigo in 3, ataxia in 3, headache in 2, tinnitus alone in 2, and facial tics in 2 accounted for the rest. Vertigo was rarely an important primary symptom and, when it occurred alone, suggested Ménière's disease rather than an 8th nerve tumor.

Of considerable interest are the clinical findings referable to the cranial nerves other than the 8th reported by González (20), as well as by Grant and Spitz (21). The 1st cranial nerve was rarely involved. The 2nd cranial nerve, however, was commonly affected. Sixty-five per cent of González' series showed papilledema. The incidence was even higher in the Grant and Spitz series, with 74 per cent showing choked disks. The 3rd, 4th, and 6th cranial nerves were seldom involved. The 5th nerve, with the exception of the 8th, was most commonly affected in González' series. Eighty-six per cent of his patients showed some sensory impairment either in the face or cornea. This was also true in the Grant and Spitz series, where 92 per cent were similarly affected; the corneal reflex alone was diminished in 72 per cent. The 7th nerve was affected in 48 per cent of

González' patients. The incidence was much higher in Grant's and Spitz' series, 82 per cent having some degree of facial nerve involvement.

Almost 96 per cent of González' patients showed varying degrees of impaired hearing. Every patient in the Grant and Spitz series had impairment of hearing, and in 35 the deafness was complete.

According to Olsen and Horrax (28), tinnitus was usually noticed at about the same time as the deafness. In 4 of 42 acoustic nerve tumors reported by the latter, tinnitus preceded deafness; it was entirely absent in 38 per cent of the patients.

Of real importance in the recognition of 8th nerve tumors is the Barany test. It was positive in 36 of the 44 patients (82 per cent) in whom it was used by Grant and Spitz. It was inconclusive in 6 patients, and in 2 it was entirely negative.

The fact that vestibular function may be little or not at all affected by the occasional 8th nerve tumor suggested that some tumors may originate in the cochlear branch rather than in the vestibular nerve.

The remaining cranial nerves were found to be involved far less frequently. The 9th and 10th nerves were each involved once. The 10th, 11th, and 12th nerves were involved as a group once. On two occasions the 12th nerve was involved.

The clinical picture in patients with bilateral acoustic tumors is essentially the same as that observed in unilateral disease (10, 20). In most instances, the 8th nerve syndrome on one side has preceded and outweighed findings on the other. The common occurrence of these tumors with von Recklinghausen's neurofibromatosis, and in rare instances meningiomas, often makes the diagnosis difficult.

ROENTGEN MANIFESTATIONS

The literature credits Henschen (23, 24) with being the first to demonstrate 8th nerve tumors radiographically. His patients were so positioned that the internal auditory meatus was projected into the external meatus, a technic which proved to be extremely difficult and not too reliable. According to Ebenius, G. Forssell verified Henschen's observations in 1911 (14).

When Cushing wrote his excellent book on acoustic nerve tumors in 1917, Henschen's technic was still being used, with far from satisfactory results, by Carr (6), who tried to popularize the method. It is interesting to note that when Schüller published his book *Roentgen Diagnosis of Diseases of the Head* in 1918 (32), it contained six pages dealing with acoustic tumors, of which but six or eight lines were devoted to Henschen's lateral skull technic and findings. In Schüller's hands it had proved unsuccessful. Even more interesting was his statement: "The pathognomonic change (the thinning and the forward bending of the dorsum sellae) is a valuable sign, making certain the diagnosis of acusticus tumor."

It was not until 1926, when Towne published his paper on *Erosion of the Petrous Bone by Acoustic Nerve Tumors* (36), that roentgen technics began to assume some stature in the recognition of these tumors. To quote Towne: "Dr. W. E. Chamberlain of the Department of Roentgenology of Stanford University Hospital has recently succeeded in showing the destruction of the petrous bone in a very fine case of acoustic nerve tumor. Instead of making lateral projections of the skull in which the internal and external auditory meati of the two sides were identified with difficulty, he used a postero-anterior projection in which the petrous bones are shown in profile. The two bones can thus be compared in one set of films. With the patient on his back, the head is flexed at the top of the neck so that the chin is drawn backward. The center of the film is placed under the foramen magnum and the central ray is directed at the center of the film through the midline of the frontal region at a point about 3 inches above the level of the eyebrows. In all three cases of verified acoustic nerve tumor examined roentgenographically with the occipital projection described in this paper, extensive erosion of the petrous bone on the side of the tumor has been

shown clearly. It seems probable that the use of this technic during the period when only auditory symptoms are present may lead to earlier diagnosis and surgical treatment, which would certainly be valuable as confirmatory evidence in the diagnosis of more advanced acoustic tumors."

Towne's article excited much favorable discussion. It was not long thereafter before the position, which Towne himself gave Dr. W. E. Chamberlain credit for developing, became known as the "Towne" position. It would seem that if this position, which Chamberlain (7) prefers to call the "occipital view," were to bear a proper name it might justifiably be called the "Chamberlain-Towne" position, as suggested by Sante (31).

Lysholm (26) was probably the first to dedicate an entire article to the standardization of roentgen technics for study of the petrous pyramid. His excellent illustrations, published in 1928, may still be referred to profitably.

The importance of carefully controlled radiographic technic in the diagnosis of 8th nerve tumors cannot be overemphasized. There are no substitutes for a fine focus tube, proper positioning, absolute immobilization of the head, and careful film processing. Furthermore, the head must be examined in every conceivable projection, as there is no one view in which the internal auditory canals are best seen all of the time.

It was Ebenius' (14) opinion that the two views most suitable for the recognition of 8th nerve tumors are the "frontal-dorsal and axial views." (His illustrations indicate that the "frontal-dorsal" view is the ordinary "occipital" position; the "axial" view the "base" or "Hirtz" position.)

Camp and Cilley (5) favored an anteroposterior position, in which the petrous pyramids are cast through the orbits. They preferred it to the basal and occipital views because it avoided superimposed confusing shadows.

In reviewing the roentgenograms of the patients included in our series of 8th nerve tumors, we attempted to determine which views of the head gave us most information. The occipital view proved slightly superior to the routine postero-anterior view in which the petrosae are projected into the orbits.

To summarize briefly, in the present series the occipital view was most informative in 48 per cent of the cases; the postero-anterior view in 37 per cent; the Stenvers position in 8 per cent, and the Hirtz position in 7 per cent. Whereas 85 per cent of the tumors were recognized most readily in the anteroposterior and postero-anterior projections, it must be borne in mind that in 15 per cent of our series, the diagnosis might have been missed had a complete examination not been done.

We routinely examine patients suspected of having angle tumors in both lateral, postero-anterior, occipital, base, and Stenvers projections. Stereoscopy is used in almost all cases, and body-section roentgenography whenever the findings are questionable.

Lateral View: The lateral projection was extremely valuable. Whereas it rarely helped localize the acoustic tumor, the fact that it was abnormal sharpened one's visual acuity. The lateral view revealed manifestations of increased intracranial pressure in 85 per cent of this series. In the entire group of 122 patients, there were but 15 in whom the lateral film was considered normal.

The hypophyseal changes will be considered below. Suffice it to say that Schüller's "pathognomonic thinning and forward bending of the dorsum sellae" were rarely seen.

Occipital View: The occipital view was uniformly reliable, though not dramatically superior to the postero-anterior. It was excellent for disclosing erosions of the postero-superior surface of the pyramid.

Postero-anterior View: Although statistically the postero-anterior view did not show itself to be as informative as the occipital view, there were many patients in whom it was the only view that showed the lesion. It has all the advantages of the occipital projection except for the fact that

the bones of the face are superimposed upon the canals. Actually, this is the best position for showing early erosions of the floor of the canal.

It is noteworthy that several patients revealed increased vascularity of the ipsilateral half of the frontal bone, a finding of questionable significance.

Stenvers View: Whereas the Stenvers projection commonly showed the effects of the 8th nerve tumor, it seldom demonstrated the bone changes to best advantage. It cannot be neglected, however, as there were several patients in whom it furnished the only reliable localizing roentgen evidence of the lesion.

Base View: The base view was the least informative of all. Whereas changes in the pori and canals could be visualized, they rarely were seen to best advantage in this projection. Occasionally, however, it afforded the only diagnostic clue, making its inclusion in routine examinations vital.

Probably of more importance than the changes in the petrosal apices were the findings in the neighboring structures. As a rule, in the base view, the foramen ovale and foramen spinosum on both sides look alike. Although minor differences may occur normally, they should always excite suspicion and lead to caution. Approximately 25 to 30 per cent of the patients in this series revealed significant changes in the appearance of these foramina. In some only the slightest atrophy of the rims was seen. In others, the foramina were larger than normal, and in a few they could hardly be visualized.

Occasionally, the jugular foramen was seen to be unusually prominent and even large on the side of the tumor. Whereas the significance of this observation was doubtful, one wondered whether it could be related to pressure of the growth upon the lateral sinus and internal jugular vein or upon the foramen itself (29).

In several instances some demineralization of the sphenoidal ridge was found on the side of the tumor. On one occasion a calcification was seen in the base view which was not seen in the other views.

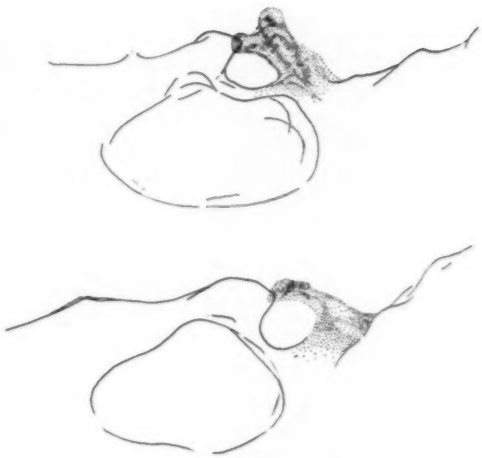

Diagram I. Sketch of the hypophyseal fossae illustrated in Fig. 22, which shows more clearly Schüller's "forward bending of the dorsum sellae."

Planigraphy: In no instance in this series did the planigram reveal changes in the internal auditory canal that had not been suspected previously from the study of the routine films. True it is, however, that the body-section technic often showed the bone defects more clearly. The procedure should always be used when routine exposures fail to demonstrate an angle tumor suspected clinically.

We have already indicated that this report is based upon our findings in 122 proved 8th nerve tumors in but 70 of which were the roentgenograms available for study. Reviewing the radiological reports contained in the records of these 122 patients, we learned that a correct preoperative diagnosis of angle tumor had been made in only 76, or 63 per cent. Our review of the 70 films still available for investigation gave better results. In 59, or 84 per cent, we thought we could see definite roentgen evidence of an angle tumor. In 4 additional cases we thought petrosal changes were present, but these were not included because we felt we had been prejudiced in favor of the diagnosis by what we knew about the case.

The literature contains somewhat similar data. Ebenius (14) reported a diagnostic accuracy of 80 per cent in acoustic tumors.

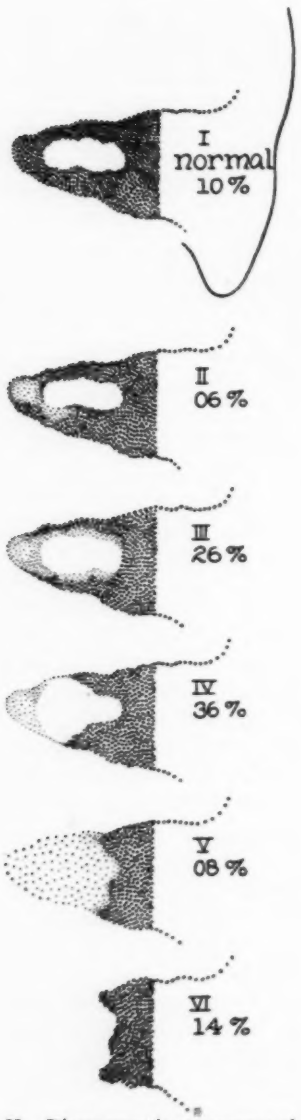

Diagram II. Diagrammatic appearance of the internal auditory canals and petrosal apices observed in patients with 8th nerve tumor. The grouping is based upon the appearance of the petrous pyramid in the occipital or postero-anterior projection. The incidence in each group is indicated by the percentage figure that accompanies the various diagrams.

In Nielsen's (27) series the proper diagnosis was made in 81 per cent, and in Bager's (2) in 80 per cent. The results were not as good in González' series (20), where the tumor was localized in the angle in but 50 per cent of the cases. It is noteworthy that the occipital position had not been employed routinely in this latter series.

When acoustic tumors produce bone changes that can be seen radiographically, they are probably due to the combined effects of direct bone pressure and indirect pressure upon the aqueduct of Sylvius. The former may be observed with any cerebellopontile angle tumor; the latter are less specific. The roentgen findings attributable to increased intracranial pressure have been repeatedly described in the literature (29). In this series thinning of the bones of the calvaria, wide sutures (both rather unusual in 8th nerve tumors), and changes in and around the hypophyseal fossa were classical.

The hypophyseal changes varied considerably. In approximately half the patients the fossa was considered enlarged. More common were double hypophyseal floors and erosions of the floor, which occurred in about two-thirds of the series. About 60 per cent revealed early atrophy to frank destruction of the dorsum sellae of the type often seen as a result of third ventricle pressure. The posterior clinoids were affected about twice as often as the anterior clinoids or tuberculum sellae (64 per cent compared to 31 per cent). In but 15 patients, of this series of 122, were the hypophyseal fossa and its environs considered normal (12 per cent). It is noteworthy that in each instance, however, there were changes in the pori acustici that suggested intracranial disease.

As the films of these 8th nerve tumor patients were reviewed, a deliberate attempt was made to evaluate the significance of Schüller's "thinning and forward bending of the dorsum sellae." We found only two fossae which seemed to parallel his description and illustrations (Fig. 22, Diagram I). Whether these are significant changes or whether they are anatomical variants distorted by the effects of increased intracranial pressure must remain an unsettled question until a greater experience has accumulated.

The petrosal bone defects merit detailed discussion. Probably as a result of direct

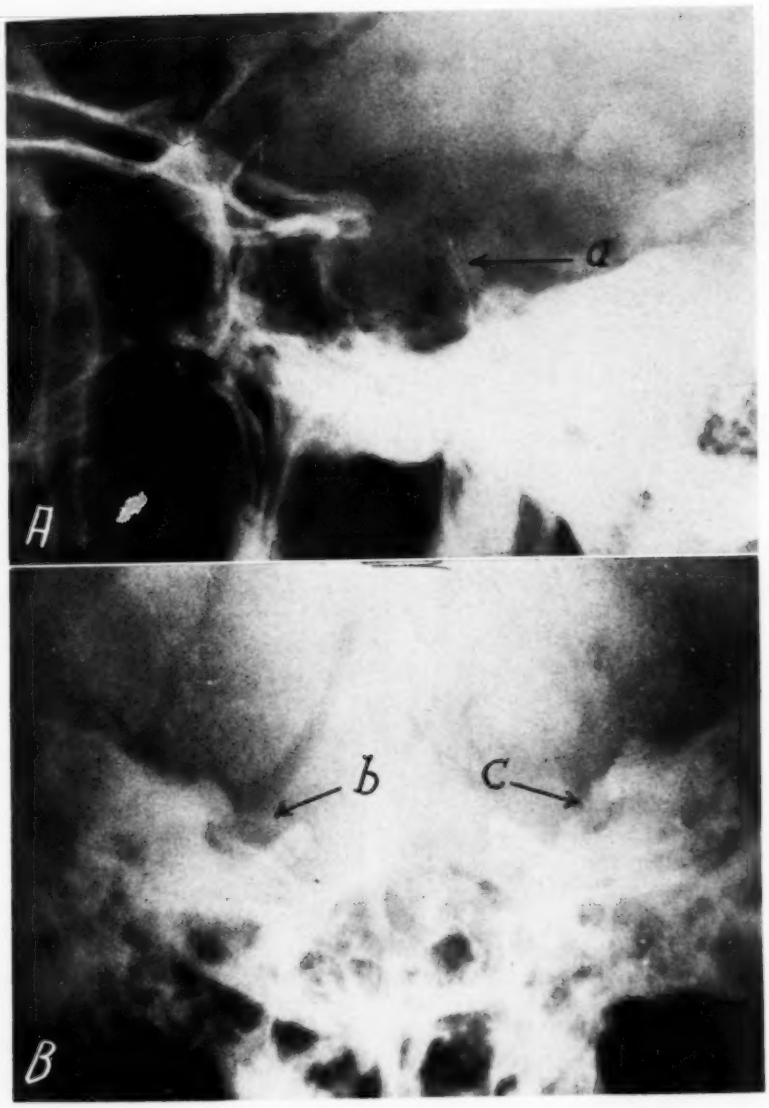

Fig. 8, A and B. Case I (Group I). Large right acoustic neurinoma.
A. Lateral view. The hypophyseal fossa is markedly eroded. The dorsum sellae is barely perceptible at *a*.
B. Occipital view. Normal and symmetrical internal auditory canals are seen at *b* and *c*. See also Fig. 8C.

pressure by the tumor, perhaps aided by pulsations from the anterior inferior cerebellar artery, veins, and neighboring sinuses (29), erosions of the internal auditory canal and petrous apex may be found in association with any cerebellopontile angle tumor. We have seen them with cholesteatomas, hemangioblastomas, meningiomas, myxomas, and astrocytomas, as well as in acoustic tumors. They have also been found associated with cysts arising in this region (35). In none of our cases did we ever see an abnormally narrow canal of the type reported by Brunner (4).

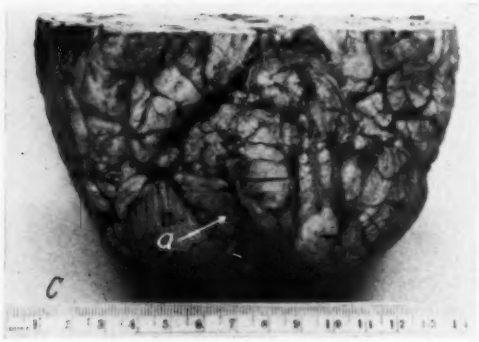

Fig. 8C. Case I. Postmortem specimen, showing huge neurinoma at *a*.

The canal changes seen radiographically in this series of 8th nerve tumors were rather characteristic. There seemed to be no consistent relationship between the size of the tumor and the degree of bone involvement, an observation also recorded in the literature (8). For purposes of description, and in order to accentuate the roentgen manifestations, we divided the findings observed radiographically in 70 patients into six groups (Diagram 2):

Group I: No roentgen abnormalities in the petrosae (10 per cent).

Group II: Slight deossification of the internal auditory canal as the only roentgen finding (6 per cent).

Group III: Wide, but not short, internal auditory canals (26 per cent).

Group IV: Short and wide internal auditory canals which in many instances were actually funnel-shaped (36 per cent). This was the largest group.

Group V: Complete destruction of the internal auditory canal, though the petrosal pyramids were still visualized radiographically, albeit considerably demineralized (8 per cent).

Group VI: Complete destruction of the internal auditory canal and the petrosal apex (14 per cent).

Group I

There were 7 patients showing no roentgen abnormalities, or 10 per cent of the series. In spite of the fact that we knew where the angle tumor had been found in these patients, we could see no evidence of it in the roentgenograms. Yet roentgen evidence of increased intracranial pressure was commonly present.

CASE I (Fig. 8): M. S., March 19, 1943. A 47-year-old woman was admitted to the hospital complaining of blindness in the left eye associated with dizziness and vomiting.

Chronology of Present Symptoms: Dizziness first occurred approximately nine months before admission. At that time, the patient noticed that her gait was unsteady. Four months before admission, her left eye began to go blind. Shortly before admission, some loss of vision was also noted in the right eye.

Positive Neurological Findings: Four diopters choking was noted in the right eye and two diopters choking in the left. The left pupil was fixed. There was some instability in the station and gait. The Barany test was negative.

Operation: A right fronto-temporal craniotomy was performed, but the tumor was not identified. The patient died and the brain was obtained for study.

Neuropathology: A large tumor was found in the right cerebellopontile angle, compressing and displacing the pons and medulla to the left side. The facial nerve was compressed only near the pons. The 8th nerve was considerably elongated and compressed around the tumor. The tumor itself weighed 23 gm. and was well encapsulated. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (March 20, 1943): The bones of the calvaria were negative. The hypophyseal fossa was definitely enlarged (anteroposterior measurement 18 mm.; depth 15 mm.). There was some erosion of the anterior clinoids and the tuberculum sellae. In addition, there was almost complete destruction of the posterior clinoids, with marked erosion of the dorsum sellae. The pineal was not calcified. No other abnormalities could be found in the skull. The internal auditory canals on both sides were perfectly normal.

Conclusion: Marked enlargement with bone destruction in the region of the hypophyseal fossa, suggesting the presence of an intrasellar tumor.

CASE II (Fig. 9): E. A., March 27, 1944. A 30-year-old white man was admitted to the hospital complaining of dizziness.

Chronology of Present Symptoms: For fifteen months, the patient had suffered from dizzy spells, which came on at irregular intervals. During this period, he had also had temporal and occipital headaches. For several months prior to admission he slept a good deal and seemed unusually forgetful.

Positive Neurological Findings: The patient was partially disoriented and his memory for recent events was poor. Some ataxia and a positive Romberg sign were noted. The left corneal reflex was considerably diminished. There was bilateral papil-

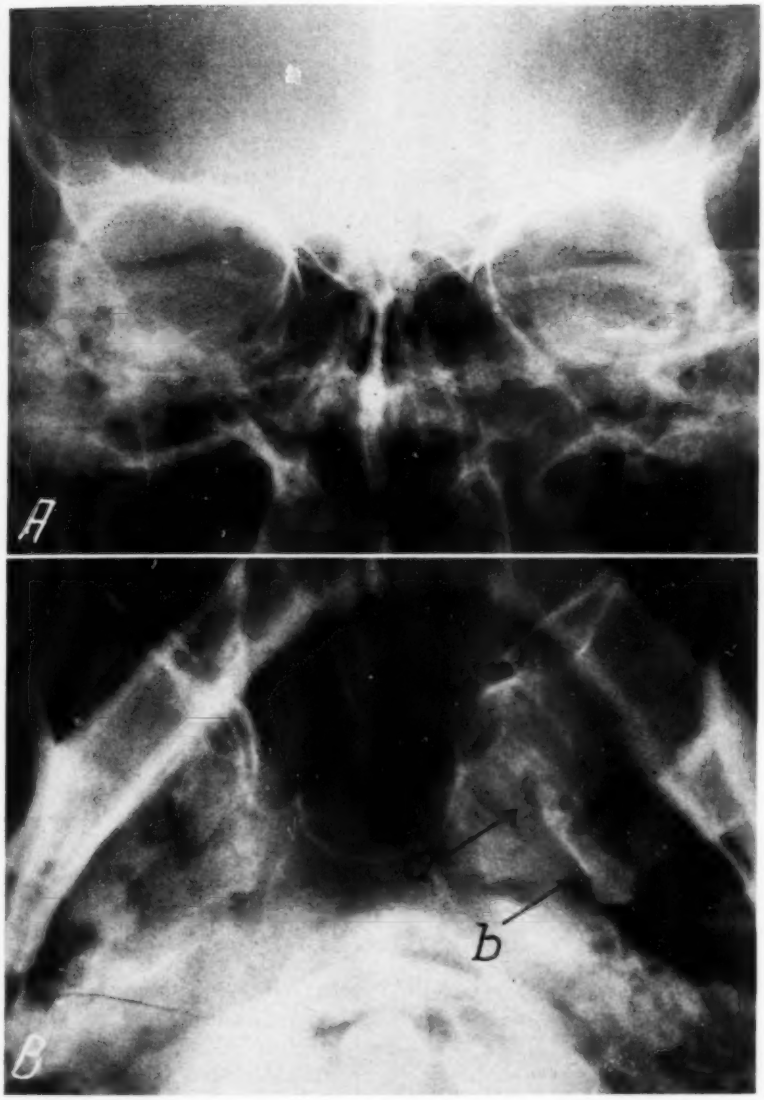

Fig. 9. Case II (Group I). Left acoustic neurinoma.
A. Postero-anterior view. The internal auditory canals are normal and symmetrical.
B. Base view. Nothing abnormal is noted. The long calcific density noted at *a* and *b* is the styloid process.

ledema with vertical and horizontal nystagmus. Slight facial weakness was noted on the left side.

Operation: A left cerebellopontile angle tumor was found, surrounded by a number of small subarachnoid cysts.

Neuropathology: The operative specimen consisted of several small fragments of tissue, the largest measuring approximately 1 cm. square. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (April 26, 1944): The bones of the calvaria were negative. The hypophyseal fossa was normal in size and shape. It was noteworthy that the posterior clinoids seemed somewhat irregular and perhaps displaced forward slightly. There was no erosion in the region of the posterior clinoids, however. The pineal was calcified and not displaced. Both internal auditory canals were beautifully visualized and seemed perfectly normal. Attention

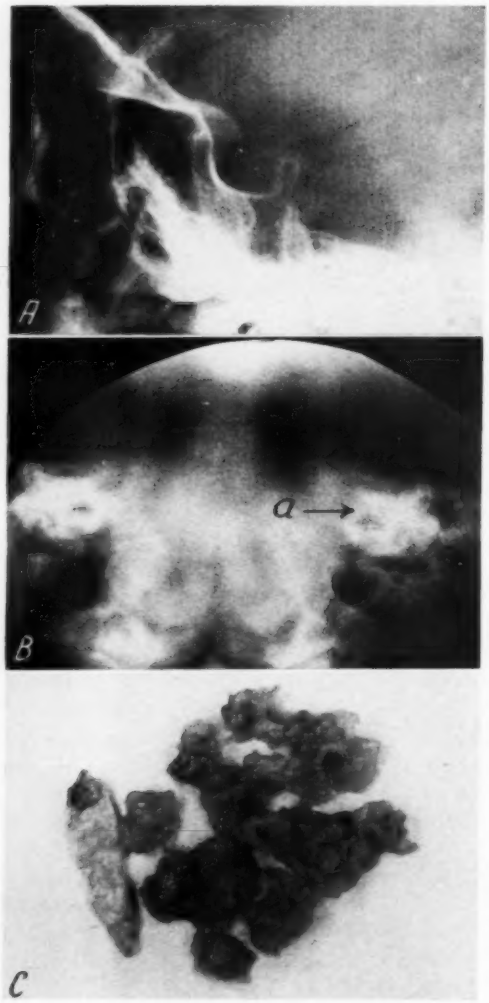

Fig. 10. Case III (Group I). Left acoustic neurinoma.
A. The hypophyseal fossa is top-normal in size.
B. Body-section study of the internal auditory canals reveals no change in the region of the left porus, *a*, where the tumor was found.
C. Portions of the tumor removed at operation.

was called to the presence of an area of calcification apparent in the Hirtz view of the base of the skull, lying in the middle fossa on the left side, obscuring the foramen ovale and foramen spinosum.[1]

Conclusion: Calcification in the middle fossa on the left side, of uncertain significance.

CASE III (Fig. 10): M. K., April 27, 1943. A 58-year-old man was admitted to the hospital complaining of progressive loss of hearing in the left ear, and dizziness.

Chronology of Present Symptoms: The patient first noticed progressive deafness in the left ear approximately two years before his admission. Eight months prior to his admission, he became dizzy. The dizziness increased rapidly and was associated with considerable difficulty in walking at the time of his hospital admission.

Positive Neurological Findings: The eye grounds revealed bilateral papilledema measuring 3 diopters. The left corneal reflex was absent. Horizontal nystagmus was present. Dysmetria and adiadochokinesia were noted in the left hand. The left 3rd, 6th, and 7th cranial nerves revealed definite weakness. The Romberg sign was positive, the patient falling to the right side.

Operation: A soft, avascular tumor was identified and partially removed from the left cerebellopontile angle.

Neuropathology: The operative specimen consisted of several pieces of soft tissue which weighed approximately 10 gm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (April 6, 1943): The bones of the calvaria were negative. The hypophyseal fossa was "top-normal," with an anteroposterior measurement of 12 mm. and depth 9 mm. No evidence of an intracranial lesion was found. Body-section films were made through both temporal bones and these also revealed no abnormalities in the region of the internal auditory canal.

Conclusion: Findings negative.

Group II

There were 4 patients showing slight deossification of the wall of the internal auditory canal, 6 per cent of the series. The probabilities are that most of these cases will continue to be missed because the roentgen changes are so indefinite. Given excellent radiographs and a high index of suspicion, it should be possible to recognize some of this group.

CASE IV (Fig. 11): R. D., Nov. 8, 1939. A 60-year-old woman was admitted complaining of pain in the occipital portion of the skull.

Chronology of Present Symptoms: Occipital headaches first occurred approximately one year before admission to the hospital. Soon thereafter, dizziness developed, with a tendency to fall toward the right side. Approximately seven months before admission, diplopia was first noticed. This was soon followed by increasing deafness in the left ear, associated with tinnitus. Shortly before admission to the hospital, numbness of the left side of the face developed.

Positive Neurological Findings: The left side of

[1] This was an error. The density seen in the middle fossa was actually due to the styloid process.

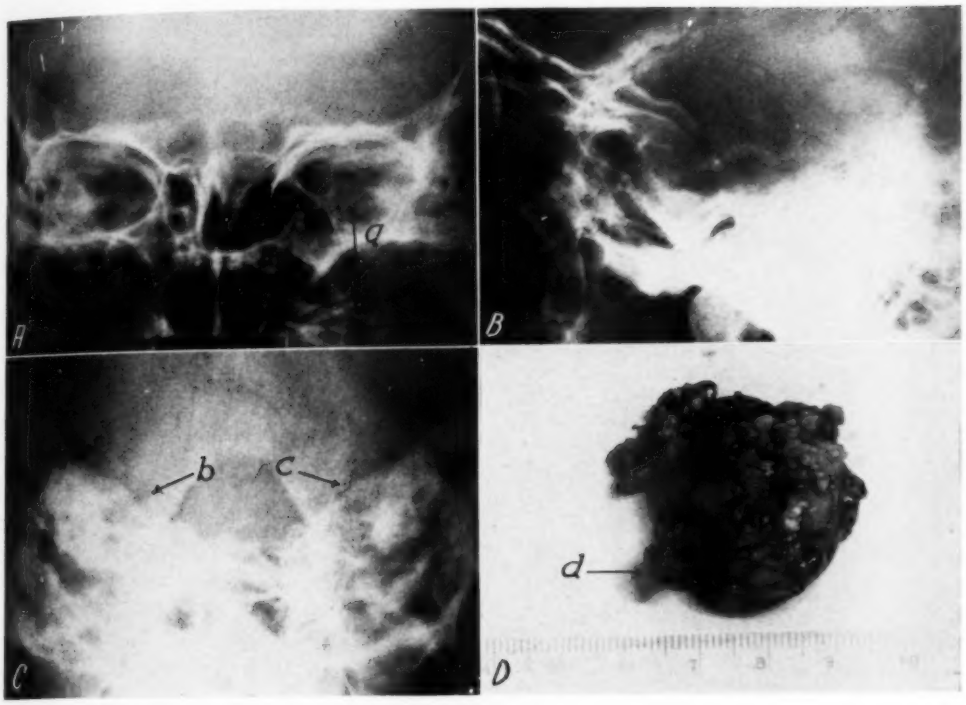

Fig. 11. Case IV (Group II). Left acoustic neurinoma.
A. Postero-anterior view. The left internal auditory canal reveals just the slightest demineralization of its inferior border, at *a*.
B. Lateral view. The hypophyseal fossa and dorsum are markedly distorted.
C. Occipital view. No changes are seen in the internal auditory canals at *b* or *c*.
D. Tumor removed at operation. Note the nipple of tissue that marks the site of the porus acusticus at *d*.

the face was numb. Spontaneous nystagmus was present when the patient looked to the right and to the left. The Barany test revealed evidence suggesting a tumor in the left cerebellopontile angle. The left ear was totally deaf. There were bilateral choked disks measuring 5 diopters. The left corneal reflex was diminished and there was some paralysis in the left external rectus muscle. The tongue deviated toward the right.

Operation: A small tumor was found in the left cerebellopontile angle and was removed for the most part. A small amount of tumor tissue was left in the internal auditory canal.

Neuropathology: The specimen consisted of a well encapsulated tumor, into which the 8th nerve entered. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Nov. 9, 1939): The bones of the calvaria were normal. The hypophyseal fossa was considerably distorted and somewhat enlarged, the anteroposterior measurement being 14 mm. and the depth 12 mm. The anterior clinoids were normal, but the tuberculum sellae seemed eroded. There was considerable erosion of the posterior clinoids and definite erosion, also, of the dorsum sellae. The pineal was calcified and not displaced. The internal auditory canals were beautifully visualized on both sides, and seemed essentially normal. There was the slightest amount of demineralization of the region of the internal auditory canal on the left side.

Conclusion: Considerable erosion and distortion of the hypophyseal fossa due to increased intracranial pressure, with the slightest amount of demineralization of the apex of the left petrous pyramid.

Group III

There were 19 patients, 26 per cent of the series, with wide but not short internal auditory canals. The fact that the canals were wide, but not short, suggested that the growths may have originated within the canal and grown medially. As a rule, one noted associated demineralization of the petrosal apex.

CASE V (Fig. 12): R. T., Jan. 11, 1944. A 51-year-old woman was admitted to the hospital complaining of noises in the head associated with weakness in the right arm and right leg.

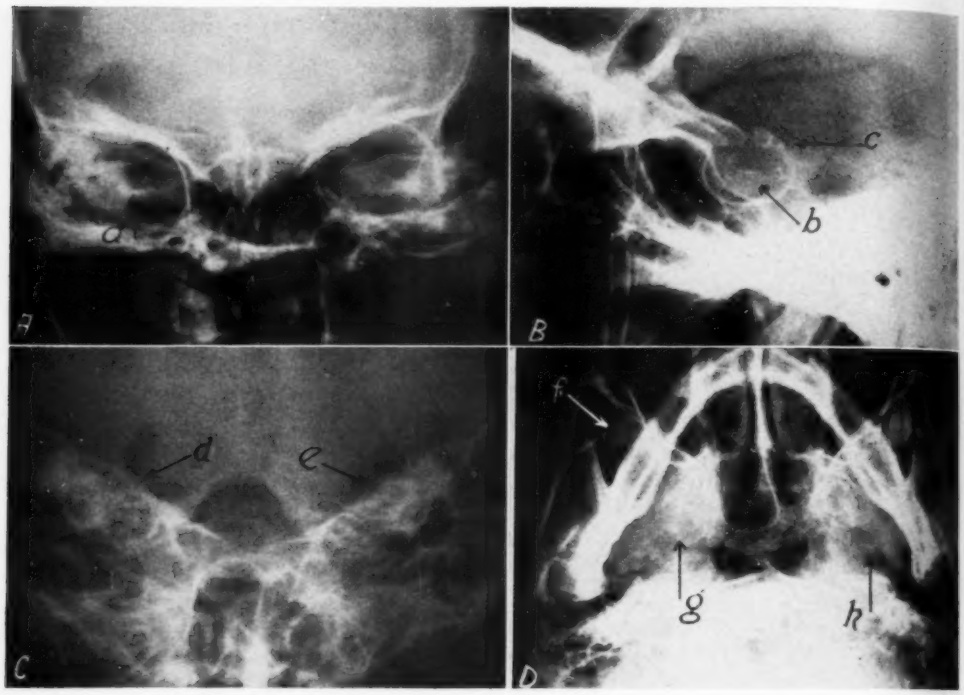

Fig. 12, A–D. Case V (Group III). Right acoustic neurino na.

A. Postero-anterior view. The right internal auditory canal, *a*, is wider than the left.

B. Lateral view. The hypophyseal fossa is slightly enlarged. The posterior clinoids seem slightly eroded, *c*. A faint calcification is noted at *b* which may be the internal carotid artery.

C. Occipital view. The internal auditory canals, *d* and *e*, are symmetrical and normal.

D. Base view. The right foramen ovale is poorly visualized at *g* compared to the normal seen at *h*. Note the slight demineralization of the right sphenoidal ridge seen at *f*. See also Fig. 12E.

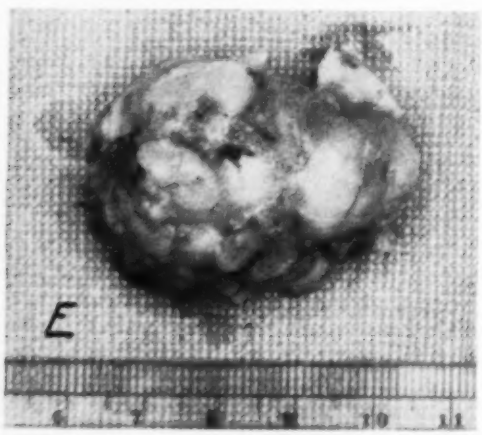

Fig. 12E. Case V. Neurinoma removed at operation

Chronology of Present Symptoms: The patient had been well until two years before admission, when she first experienced some loss of hearing and tinnitus in the right ear. One year later, she had difficulty in walking, particularly marked in her right leg. Recently, she had complained of vertigo and blurred vision.

Positive Neurological Findings: The gait was ataxic and the patient was unable to stand without support. There was moderate weakness of the right side of the face. Dysergia, dysmetria, and adiadochokinesia were noted on the right side. The 5th, 7th, 8th, and 9th cranial nerves were weak.

Operation: A tumor was found in the right cerebellopontile angle and was almost entirely removed.

Neuropathology: The specimen consisted of a lobulated encapsulated tumor measuring 4.0 × 2.5 cm. It weighed 19 gm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Jan. 13, 1944): The bones of the calvaria were negative. The hypophyseal fossa was definitely enlarged (anteroposterior measurement 14 mm.; depth 12 mm.). The right anterior clinoid seemed slightly eroded. A double floor was evident in the base of the hypophyseal fossa. The posterior clinoids were slightly eroded and seemed to

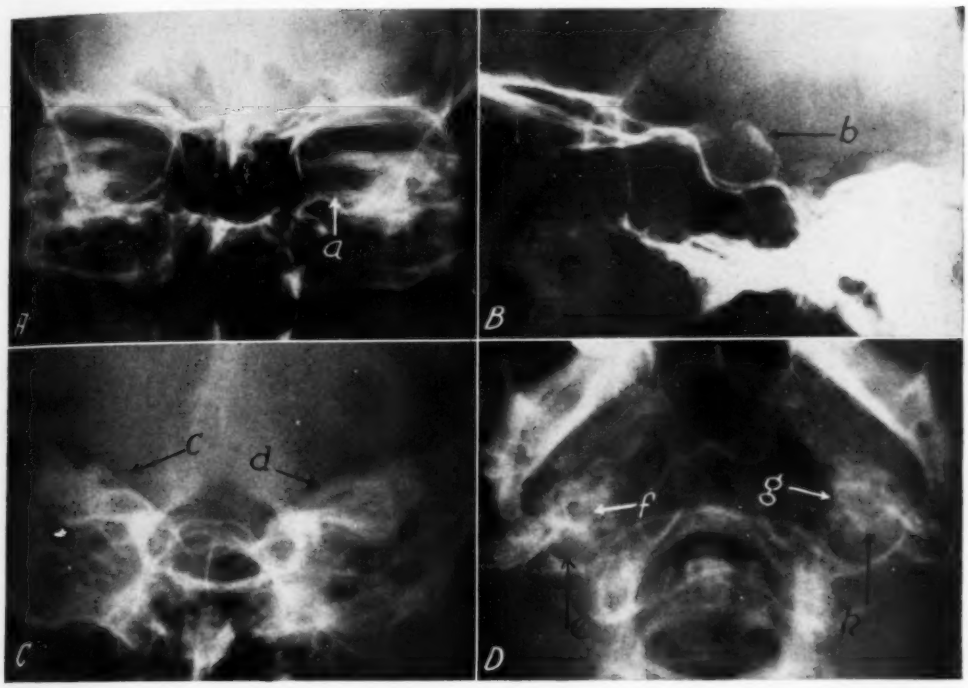

Fig. 13. Case VI (Group III). Left acoustic neurinoma.
A. Postero-anterior view. The left internal auditory canal, *a*, is wide but not shortened.
B. Lateral view. The hypophyseal fossa is normal in size and shape. There seems to be a little atrophy of the dorsum sellae at *b*.
C. Occipital view. The left internal auditory canal, *d*, is slightly dilated compared to the normal right at *c*.
D. Base view. The internal auditory canals, *f* and *g*, seem symmetrical. The left jugular foramen, *h*, is larger than the right, *e*.

be pushed forward. There was some atrophy of the dorsum sellae. Films of the base of the skull failed to visualize the foramen ovale in the middle fossa on the right side as clearly as the normal appearing left foramen ovale. In addition, a little demineralization of the sphenoidal ridge on the right side was noted in the view of the base of the skull. The anteroposterior films revealed both internal auditory canals clearly, but that on the right side was somewhat wider than the one on the left. The length of the two was comparable. The porus acusticus internus on the right side was also larger than that on the left. Some calcification was present in the region of the hypophyseal fossa which could be due to calcification in one of the internal carotid vessels.

Conclusion: Changes in the region of the internal auditory canal and middle cranial fossa on the right side compatible with a cerebellopontile angle tumor.

CASE VI (Fig. 13): A.C., Nov. 26, 1944. A 47-year-old white female was admitted complaining of tinnitus and deafness in the left ear.

Chronology of Present Symptoms: Eighteen months before admission, the patient first noticed that she was becoming deaf in her left ear. The deafness was soon associated with tinnitus. Headaches developed shortly thereafter, more marked on the left side. Increasing difficulty in walking followed, until the patient began to stagger and lurch. Two months before admission to the hospital, partial paralysis of the left side of the face developed.

Positive Neurological Findings: A definite peripheral facial palsy was noted on the left side. The Romberg test was positive, the patient falling to the left. Approximately 40 per cent loss of hearing was noted in the left ear. The Barany test indicated the presence of a left cerebellopontile angle tumor.

Operation: A large tumor was found springing from the left internal auditory canal, which was thought grossly to be a meningioma.

Neuropathology: The specimen consisted of several large portions of cerebellar tissue and smaller portions of tumor. The latter weighed 2.5 gm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Nov. 27, 1944): The bones of the calvaria were negative. The hypophyseal fossa was normal in size and shape. One had the impression that the posterior clinoids were pushed forward

Fig. 14. Case VII (Group III). Left acoustic neurinoma.
A. Postero-anterior view. The left internal auditory canal, *a*, is dilated but not short.
B. Lateral view. The dorsum sellae is considerably eroded. The hypophyseal fossa is not enlarged.
C. Acoustic neurinoma removed at operation.

slightly. There was the slightest amount of atrophy of the dorsum sellae. The internal auditory canals differed slightly on the two sides: the left internal auditory canal was a little wider than the right and the porus acusticus internus was a little wider than that on the right side. A slight amount of demineralization was seen at the apex of the left petrous pyramid.

Conclusion: Minimal changes in the region of the left internal auditory canal compatible with an 8th nerve tumor on the left side.

CASE VII (Fig. 14): M. W., June 22, 1945. A 27-year-old woman was admitted to the hospital complaining of loss of vision, headaches, and vomiting.

Chronology of Present Symptoms: Headaches and vomiting first occurred approximately a year and a half before admission to the hospital. Soon thereafter, the patient began to have trouble walking. For at least one year, she had noticed tinnitus in the left ear. Approximately two months before admission, blurred vision and diplopia developed. Vomiting first occurred about a week before admission.

Positive Neurological Findings: There was approximately 6 diopters choking in each eye. The Romberg test was positive. The gait was ataxic and the patient staggered to the left. The left hand and leg revealed dyssynergia, dysmetria, and adiadochokinesia.

Operation: A large tumor was partially removed from the region of the posterior foramen lacerum on the left side.

Neuropathology: The specimen consisted of a round encapsulated lobulated mass measuring 4.0 × 3.0 × 2.5 cm. It weighed approximately 25 gm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (June 23, 1945): The bones of the calvaria were normal. The hypophyseal fossa was not enlarged. There was marked atrophy of the dorsum sellae, with almost complete obliteration of the posterior clinoid processes. The pineal was calcified but not displaced. There was a definite difference in the appearance of the internal auditory canals on the two sides: the left was definitely wider than the right, and the left porus acusticus internus was wider than that on the normal right side.

Conclusion: Changes in the region of the left internal auditory canal compatible with an 8th nerve tumor.

Group IV

Group IV, the largest, included 25 patients, 36 per cent of the total. One usually had little difficulty recognizing the short and abnormally wide internal auditory canal, which in many was actually funnel-shaped (Figs. 16 and 17). Demineralization of the apex of the petrosa was also found. It is only fair to state that occasionally one had trouble deciding whether a patient fell into Group III or Group IV.

CASE VIII (Fig. 15): C. H., March 7, 1947. A 59-year-old female was admitted to the hospital complaining of tinnitus and loss of hearing in the right ear.

Chronology of Present Symptoms: The patient had first noticed progressive loss of hearing in the right ear approximately five years before admission.

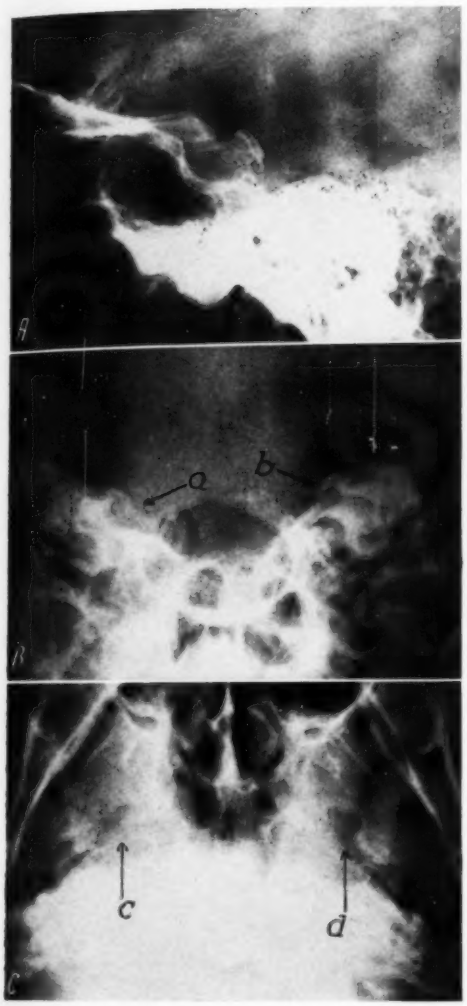

Fig. 15. Case VIII (Group IV). Right acoustic neurinoma.

A. Lateral view. The hypophyseal fossa is normal in size and shape. B. Occipital view. The right internal auditory canal, *a*, is short, wide, and funnel-shaped compared to the normal-appearing left internal auditory canal, *b*.

C. Base view. The right foramen ovale and foramen spinosum, *c*, are eroded and indistinct compared to the normal-appearing structures in the middle fossa on the left side seen at *d*. See Fig. 15, D and E.

Tinnitus, however, did not begin until about eight months prior to admission. Approximately one year before admission, difficulty in walking, with a tendency to fall toward the right side, was first observed. At about that time, the patient noticed that her right eye watered considerably. Several months before admission, severe headaches with some blurred vision occurred for the first time.

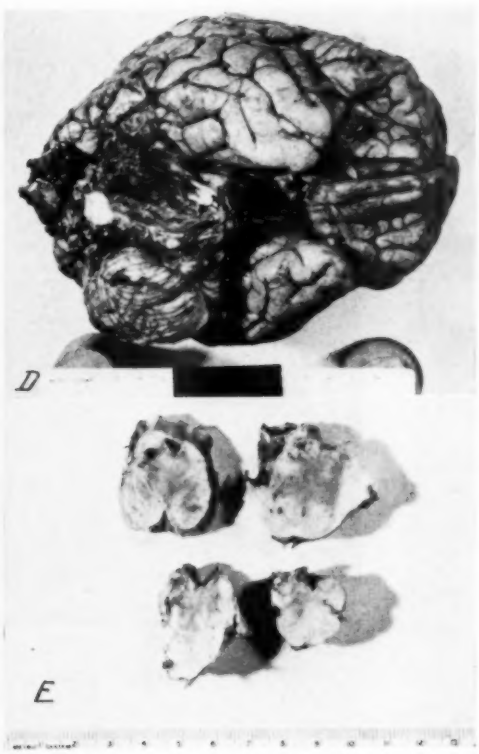

Fig. 15, D and E. Case VIII. D. Base view of the brain revealing the operative site. E. Acoustic neurinoma removed at operation.

Positive Neurological Findings: Bilateral choked disks measuring 2 diopters were observed, associated with horizontal nystagmus in both eyes. Some hypesthesia was noted in the right side of the face. The right corneal reflex was absent. Hearing was considerably impaired in the right ear. The Romberg test was positive.

Operation: A cerebellopontile angle tumor was found on the right side.

Neuropathology: The specimen consisted of a hard tumor apparently well encapsulated and granular, measuring 2.0 × 1.5 × 1.0 cm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Feb. 28, 1947): The bones of the calvaria were negative. The hypophyseal fossa was normal in size and shape, although there was the slightest suggestion of a double floor at its base. The pineal was calcified and not displaced. There was a definite difference in the appearance of the internal auditory canals on both sides: the right was shorter and wider than the normal-appearing one on the left. In addition, the films of the base of the skull revealed some impairment of visualization of the foramen ovale and the foramen spinosum on the right side.

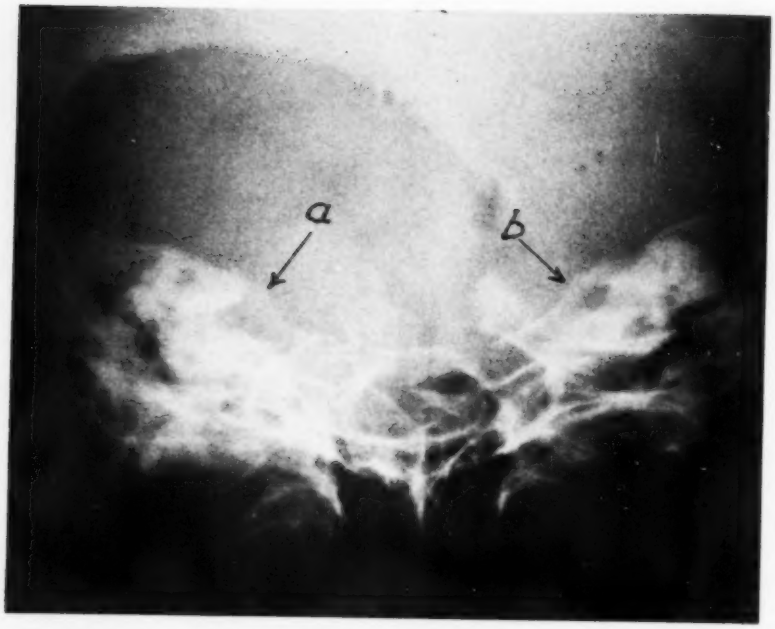

Fig. 16. Group IV. Right acoustic neurinoma. Occipital view revealing right internal auditory canal, *a*, which is short, wide, and funnel-shaped compared to the normal seen at *b*.

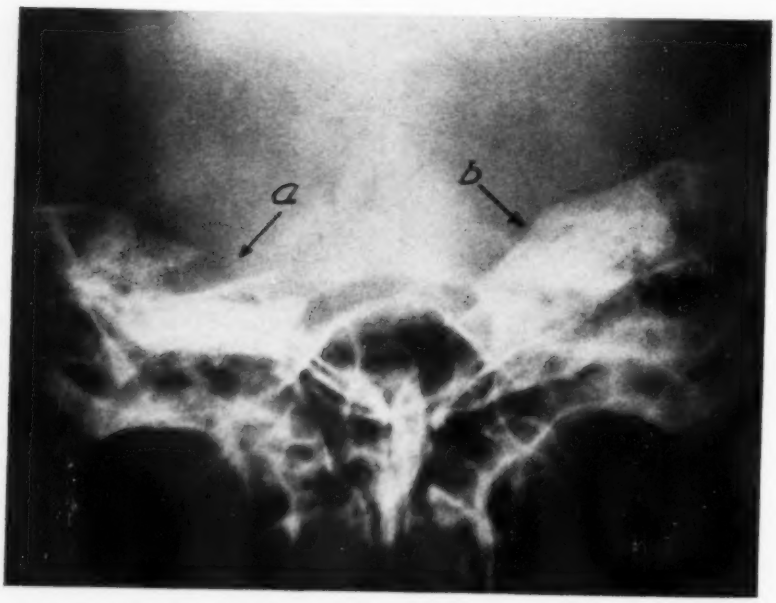

Fig. 17. Group IV. Right acoustic neurinoma. Occipital view revealing right internal auditory canal, *a*, which is short, wide, and funnel-shaped compared to the normal seen at *b*.

Conclusion: Changes in the region of the internal auditory canal on the right side compatible with an 8th nerve tumor.

Group V

There were 6 patients in Group V, 8 per cent of the series. It included the patients in whom the internal auditory canal could no longer be seen radiographically but in whom the petrous apex, though eroded, was still visualized.

CASE IX (Fig. 18): F.P., Aug. 20, 1947. A 41-year-old woman was admitted complaining of headache and difficulty in walking.

Chronology of Present Symptoms: The patient first noticed weakness and in-co-ordination in the left hand seven months prior to her admission. At that time, too, she began to realize that she was becoming deaf in her left ear. The deafness was not associated with tinnitus. Four months before admission, the patient became ataxic. Two weeks prior to admission, blurred vision was first noticed.

Positive Neurological Findings: The eye grounds revealed bilateral choked disks measuring three diopters. Hypesthesia was noted along the course of the left trigeminal nerve and to a lesser degree, along the right trigeminal nerve. There was central paresis of the 7th nerve on the left side. Hearing in the left ear was diminished. The corneal reflexes were absent on both sides. Dyssynergia and adiadochokinesis were noted in the left arm and leg. The Barany test was positive.

Operation: A cerebellopontile angle tumor on the left side was completely removed.

Neuropathology: The specimen consisted of a large mass and numerous small pieces of tumor, together weighing 40 gm. The large mass measured 4.0 × 3.0 × 3.0 cm. *Microscopic Description:* Neurinoma.

Roentgen Findings (Aug. 17, 1947): The bones of the calvaria were normal. The hypophyseal fossa was enlarged and revealed a double floor. The anterior and posterior clinoids were eroded, and the posterior clinoids seemed pushed forward slightly. Marked atrophy of the dorsum sellae was also noted. The left internal auditory canal was entirely eroded. The apex of the right petrosal pyramid showed marked atrophy.

Conclusion: Changes in the region of the left internal auditory canal compatible with an 8th nerve tumor.

Group VI

This group included 10 patients, 14 per cent. It was the most impressive group because of the complete destruction of the petrosa medial to the internal ear. Though usually smooth, the bone erosion in some cases looked infiltrated and "chewed-up."

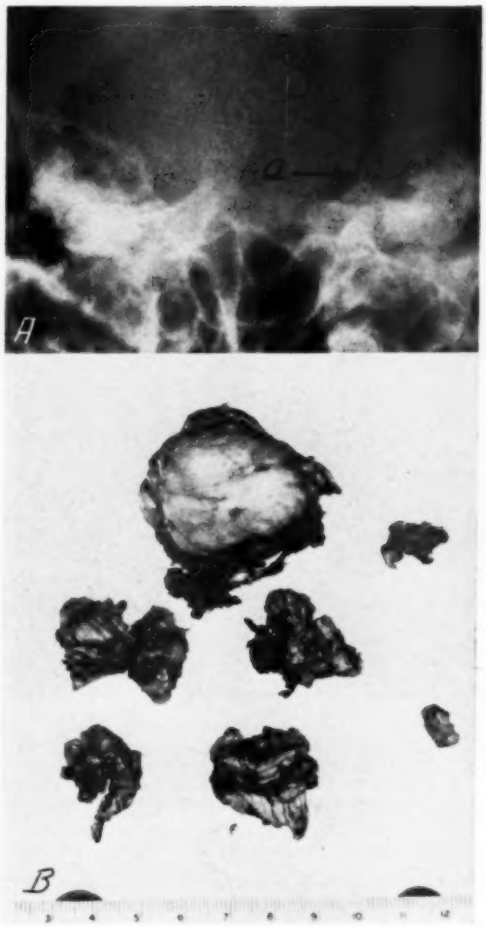

Fig. 18. Case IX (Group V). Left acoustic neurinoma.
A. Occipital view. The internal auditory canal is completely eroded, *a*, and there is definite demineralization of the apex of the left petrous pyramid.
B. Acoustic neurinoma removed at operation.

CASE X (Fig. 19): B. G., March 10, 1947. A 46-year-old female was admitted to the hospital complaining of progressive deafness in the left ear associated with lack of control of the right side of the body.

Chronology of Present Symptoms: The patient first noticed that she had poor control of her right leg, and tended to fall to the right side on walking, approximately three years before admission. Some impairment of hearing in the left ear was also noticed at this time, associated with periods of transient dizziness. Diplopia was present two years before admission. Five weeks before admission the patient fainted for the first time.

Positive Neurological Findings: Bilateral papil-

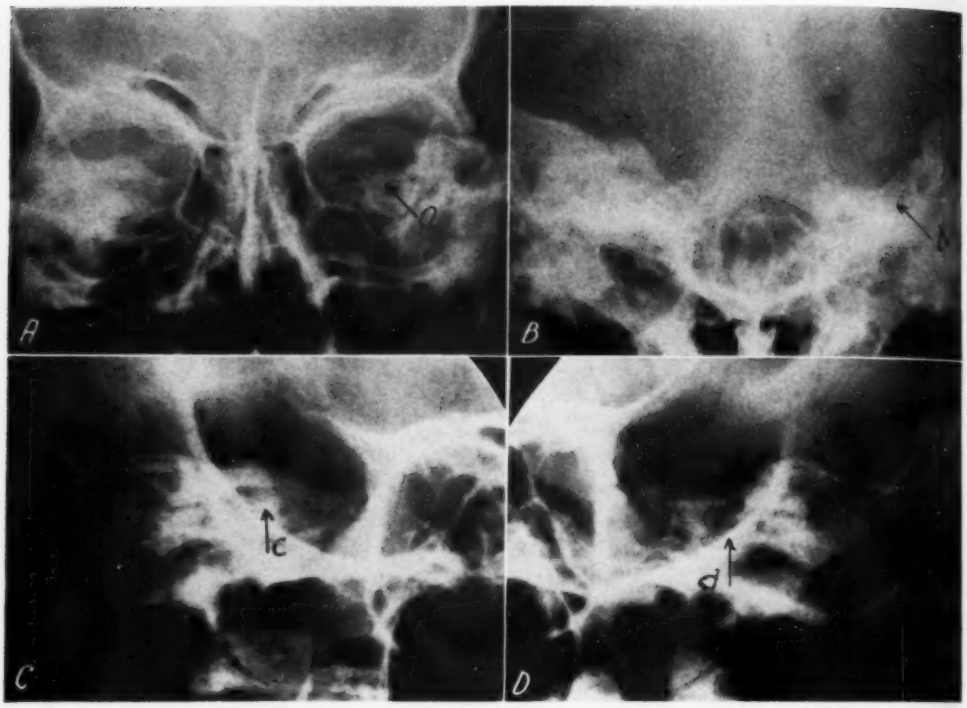

Fig. 19, A–D. Case X(Group VI). Left acoustic neurinoma.
A. Postero-anterior view. Marked destruction of the apex of the left petrous pyramid is noted at *a*.
B. Occipital view. The left internal auditory canal and petrous apex are entirely destroyed, *b*.
C. Stenvers projection. The right internal auditory canal is normal, *c*.
D. Stenvers position. There is complete destruction of the left petrosal apex and internal auditory canal, *d*.
See also Fig. 19, E and F.

ledema measuring 3 diopters was noted, associated with considerable impairment of corneal reflex in the left eye. Moderate left facial paresis was present. The left ear was deaf. There was nystagmus on looking to the right and to the left. The Romberg test was positive, the patient falling to the right. The Barany test revealed evidence of a lesion in the left cerebellopontile angle. The right arm and the left leg were ataxic. Adiadochokinesia was noted in the left hand.

Operation: A tumor in the region of the left cerebellopontile angle was exposed and partially removed.

Neuropathology: The specimen consisted of a fairly well encapsulated tumor weighing 26 gm. It measured 5.0 × 4.0 × 3.0 cm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Jan. 14, 1947): The bones of the calvaria were normal. The hypophyseal fossa was definitely enlarged (15 mm. anteroposterior measurement; 15 mm. depth). The anterior clinoids were normal, but the tuberculum sellae seemed slightly eroded. The posterior clinoids also showed some erosion and there was atrophy of the dorsum sellae. Definite destruction of the apex of the left petrous pyramid was beautifully visualized in the anteroposterior, postero-anterior and Stenvers projections. Associated with this destruction was evidence of some calcific debris, suggesting the presence of a calcified tumor.

Conclusion: Calcified mass lesion in the region of the left internal auditory canal, probably due to an 8th nerve tumor.

CASE XI (Fig. 20): M. M., Sept. 15, 1947. A 48-year-old female was admitted complaining of headache and dizziness.

Chronology of Present Symptoms: The patient first suffered from severe occipital headaches and dizziness approximately two years before admission. At that time, too, some unsteadiness of gait was observed. Soon thereafter, paresthesia of the left arm and hand and twitching of the fingers of the left hand developed. Tinnitus in the right ear followed. Approximately one year before admission, the left side of the face began to twitch and there was an increasing tendency for the left eye to close spasmodically.

Positive Neurological Findings: The Romberg test was positive, the patient falling to the left. There was total anosmia. Marked bilateral choked disks were noted. The cornea of the left eye was anesthetic.

Operation: A large vascular tumor was found in the region of the left cerebellopontile angle, extending high in the midline.

Neuropathology: The specimen consisted of two pieces of tissue, one a portion of the cerebellum and the other the tumor. The tumor tissue measured 2.7 × 2.0 × 2.0 cm. and weighed approximately 3 gm. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (Sept. 6, 1947): The bones of the calvaria were normal. The hypophyseal fossa was definitely enlarged (anteroposterior measurement 14 mm., depth 11 mm.). The anterior clinoids were normal, but there was some erosion of the tuberculum sellae. A double floor was observed at the base of the hypophyseal fossa. The posterior clinoids were considerably eroded, and there was notable atrophy of the dorsum sellae. The apex of the left petrous pyramid was markedly eroded, with complete destruction of the internal auditory canal on the left side. These changes were clearly visualized in the films of the base of the skull, as well as in those made in the postero-anterior projection and in the Stenvers position.

Conclusion: Marked destruction of the apex of the petrous pyramid associated with changes in the hypophyseal fossa compatible with an 8th nerve tumor on the left side.

CASE XII (Fig. 21): I. W., June 16, 1943. A 14-year-old male was admitted complaining of weakness in the left side of the face.

Chronology of Present Symptoms: The patient first noticed tinnitus in the left ear three years before admission to the hospital. Examination at that time revealed marked impairment of hearing in the left ear. Approximately three weeks before admission weakness of the left side of the face was first noticed.

Positive Neurological Findings: There was definite weakness of the 7th nerve on the left side, of the peripheral type. Marked impairment of hearing was noted in the left ear, and there was slight swaying toward the left side. An intention tremor was present in the left hand, with adiadochokinesia. The left corneal reflex was absent. The eye grounds did not reveal any evidence of increased intracranial pressure. The Barany test indicated a lesion in the left cerebellopontile angle.

Operation: A rather firm tumor was found in the region of the left internal auditory canal, and was easily removed except for a segment which was intimately connected to the foramen lacerum.

Neuropathology: The specimen consisted of two large pieces of tissue, both of which were rather firm and fibrous. *Microscopic Description:* Acoustic neurinoma.

Roentgen Findings (June 16, 1943): The bones of the calvaria were negative. The hypophyseal fossa was normal in size and shape. There was a suggestion of a double floor at the base of the hypophyseal fossa. A peculiar calcific streak was noted in the lateral view of the head, behind the dorsum sellae and in front of the mastoids. The sagittal views of the skull revealed marked destruction of the apex of the left petrous pyramid, with complete obliteration of the left internal auditory canal.

Conclusion: Extensive destruction of the apex of the left petrous pyramid compatible with an 8th nerve tumor.

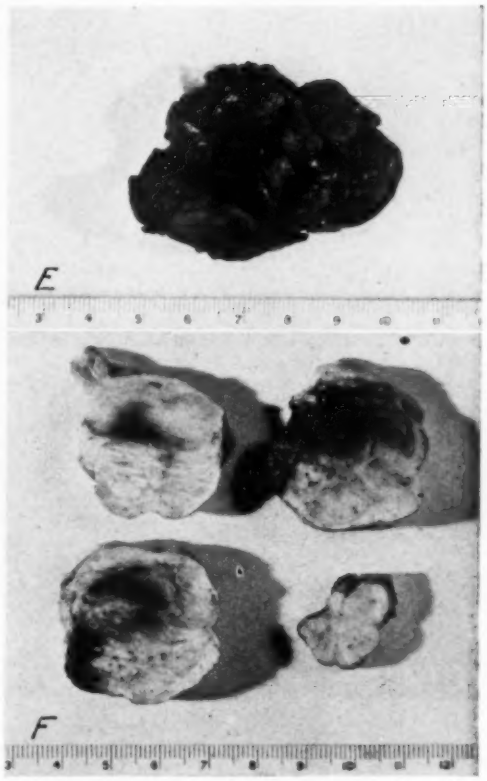

Fig. 19, E and F. Case X. E. Acoustic neurinoma removed at operation.
F. Cross-sections of the acoustic tumor revealing its fibrous and hemorrhagic character.

We should like to re-emphasize that the grouping employed here is an arbitrary one, elected because it seemed to emphasize the radiologic findings. One might postulate that all 8th nerve tumors, if left alone, would grow progressively from Group I through Group VI. Yet we have not seen this happen. Whereas it is often impossible to obtain the true growth rate of these

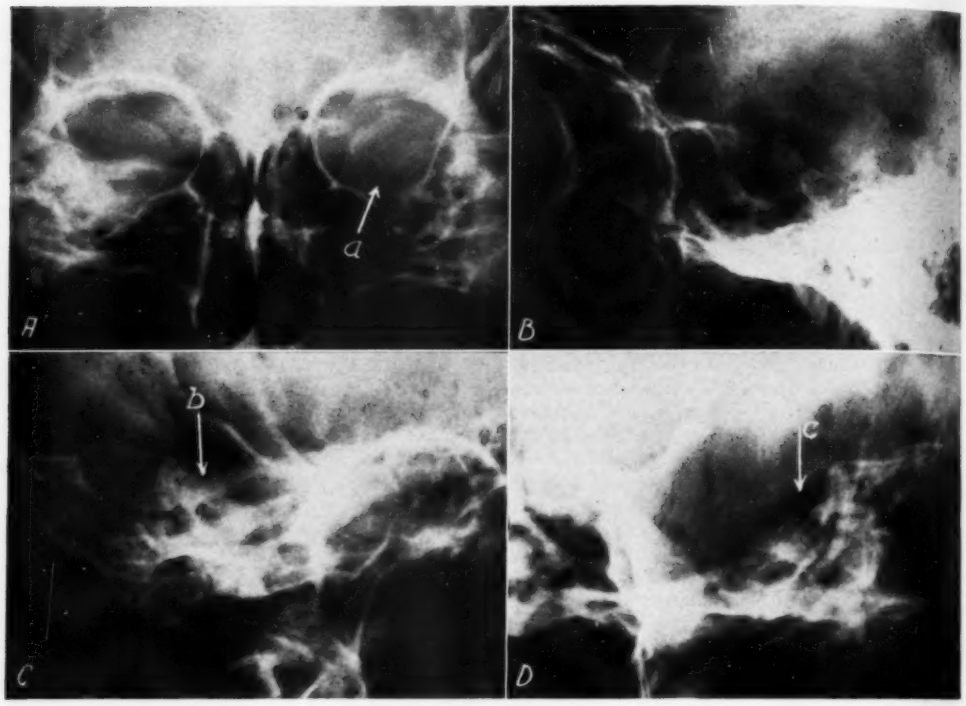

Fig. 20, A–D. Case XI (Group VI). Left acoustic neurinoma.
A. Postero-anterior view. The apex of the left petrous pyramid, *a*, is completely destroyed.
B. Lateral projection. There is marked distortion of the hypophyseal fossa with considerable erosion of the dorsum sellae.
C. Stenvers projection. The normal right internal auditory canal is seen at *b*.
D. Stenvers projection. The complete destruction of the left petrous pyramid medial to the semicircular canals is seen at *c*. (See also Fig. 20E.)

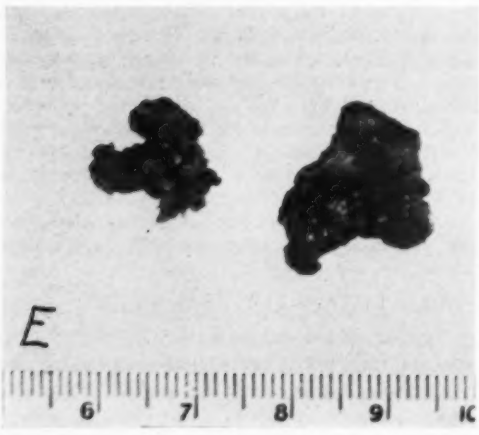

Fig. 20E. Case XI. Sections of the tumor removed at operation. A large portion of the tumor was left behind.

tumors, because they so commonly have cystic elements, most authorities agree they grow very slowly. We followed 2 cases for five years and observed no appreciable difference in the roentgen appearance. We also followed several patients with recurrences without seeing progressive petrosal erosion.

In a group of 10 patients asked to return for follow-up roentgenograms of the skull several years after having been successfully operated upon, we found no change in the appearance of the petrosae. In 2, we thought that the eroded dorsum sellae had recalcified slightly. In none had the occipital craniectomy defect filled in with bone.

We could find nothing in the literature concerning calcified 8th nerve tumors except a communication from Reeves (30). In a patient with bilateral neurofibromas, he found histologic evidence of calcium

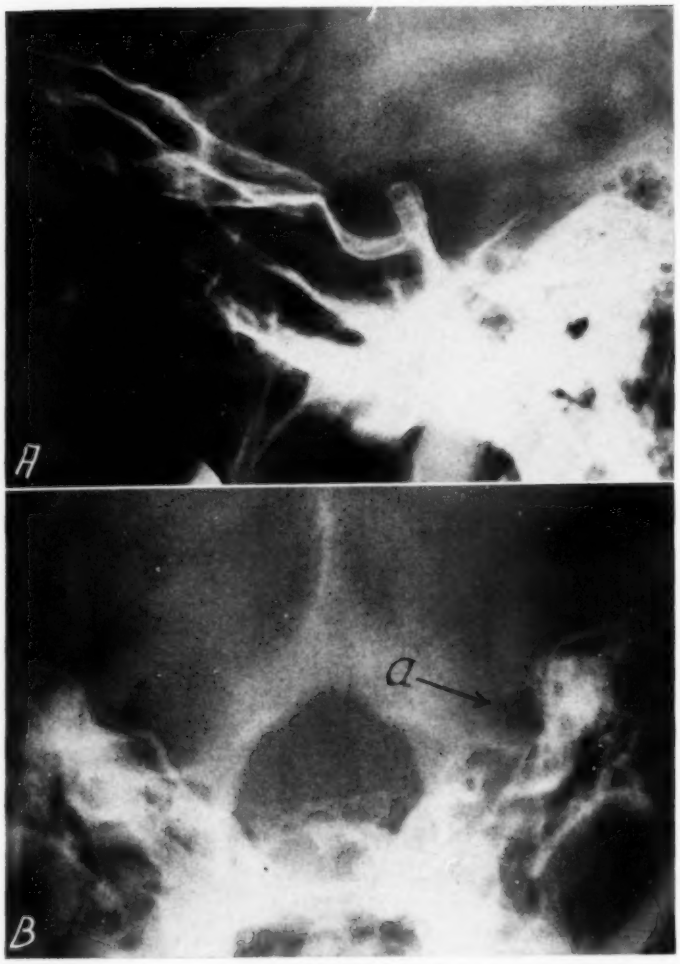

Fig. 21. Case XII (Group VI). Left acoustic neurinoma.
A. Lateral view. The hypophyseal fossa is normal in size and shape.
B. Occipital view. The left petrosal apex is completely destroyed medial to the region of the internal ear at *a*.

"in the form of small calcospherites such as are more common in meningiomas."

In the group of 8th nerve tumors reported here there were 3 showing roentgen evidence of calcification. Two were neurinomas, the third a neurofibroma (Fig. 23). It is noteworthy that the neuropathologists could find no calcific debris in these tumors microscopically, even though they were told that it was apparent in the roentgenogram. That the calcium-bearing portion of the tumor might not have been sectioned could account for this disparity. In view of the uncertainty that still exists among neuropathologists concerning these neoplasms, there is the possibility that these calcium-bearing tumors may one day be classified differently. This gains added significance when one appreciates that neurinomas and neurofibromas in other parts of the body do not become calcified.

CASE XIII (Fig. 23): A. K., Nov. 18, 1931. A 21-year-old male was admitted to the hospital complaining of increasing deafness in both ears, asso-

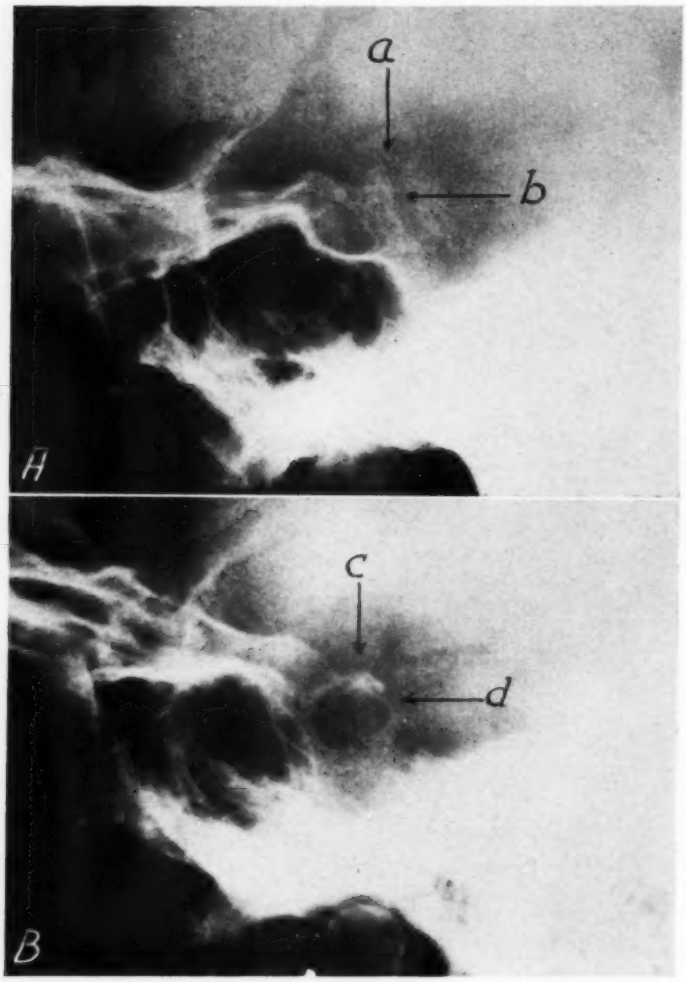

Fig. 22. A and B reveal the hypophyseal fossae of two individuals with proved 8th nerve tumors. The appearance of the dorsum in each instance is rather unusual. It suggests the changes described by Schüller as "forward bending of the dorsum sellae." The significance of this appearance is questionable.

ciated with visual impairment and a staggering gait.

Chronology of Present Symptoms: The patient first noticed impaired hearing in the right ear about two years and a half before hospitalization. This began as a humming sensation and was soon followed by increasing deafness. Approximately three months before being admitted to the hospital, the same type of humming was noted in the left ear, and this also was followed by loss of hearing. At about this time, the patient noticed that he staggered while walking, and shortly after this, dimness of vision developed.

Positive Neurological Findings: Bilateral choked disks were observed. The right corneal reflex was absent, the left considerably impaired. Nystagmus was present in both eyes on looking in all directions. The gait was extremely unsteady. Dysmetria and adiadochokinesia were noted in both hands.

Operation: The tumor was not found. The patient died.

Neuropathology: Examination of the base of the brain revealed a large tumor in the right cerebellopontile angle, measuring 5.0 × 4.0 cm. In the left cerebellopontile angle was a similar tumor, 2.0 cm. in diameter. In addition, multiple small encapsulated fibrous tumors were found along the base of

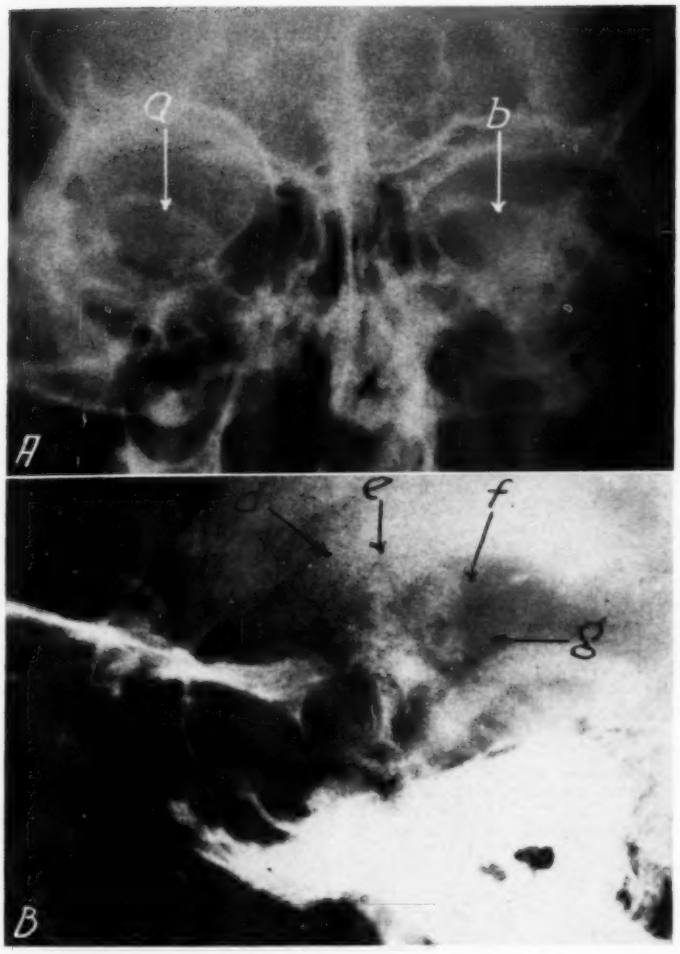

Fig. 23, A and B. Case XIII. Bilateral 8th nerve tumors due to disseminated neurofibromatosis.

A. Postero-anterior view. Both internal auditory canals are almost entirely destroyed, *a* and *b*. Both petrosal apices are demineralized.

B. Lateral view. The calcified tumor is outlined by the arrows *c* to *g* inclusive. The hypophyseal fossa is slightly enlarged and there is some erosion of the dorsum sellae. (See also Fig. 23, C and D.)

the brain, and along several of the cranial nerves. *Microscopic Description:* Multiple perineural neurofibroma.

Roentgen Findings: (Nov. 20, 1931): The bones of the calvaria showed slightly increased prominence of the digital markings, suggesting a slight increase in intracranial pressure. The hypophyseal fossa was definitely distorted and somewhat enlarged. It measured 12 mm. in anteroposterior diameter and 12 mm. in depth. The posterior clinoids seemed to be pushed forward slightly, and there were definite erosion and atrophy of the dorsum sellae. A calcified mass was observed in the region of the middle fossa, which overlay the dorsum sellae. The pineal was calcified and displaced toward the left side. There were erosion and enlargement of both internal auditory canals. The erosion was more marked on the right side than on the left.

Conclusion: Increased intracranial pressure associated with a calcified tumor in the middle fossa.

The pineal was displaced laterally in 10 individuals of the 70 whose films we reviewed. The displacement was usually but a few millimeters. We never saw the pineal

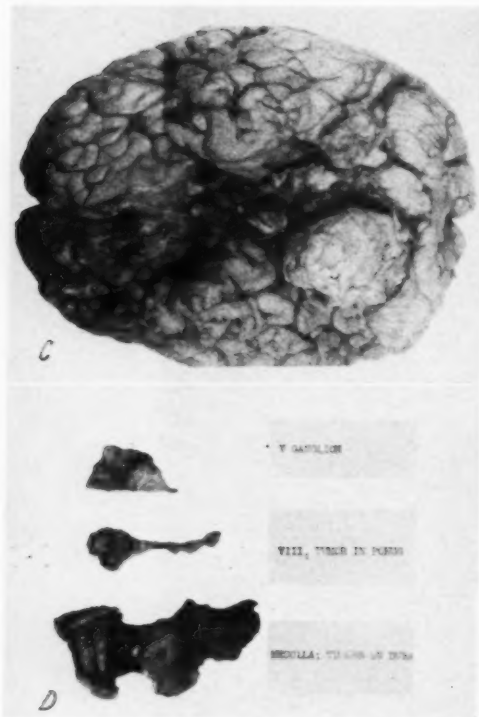

Fig. 23, C and D. Case XIII. C. Base view of the brain revealing disseminated tumor nodules extending throughout the base of the brain, with the largest nodules visualized in the region of the posterior fossa.
D. Sections showing involvement of the 5th nerve, 8th nerve, and the medulla.

pushed upward even though we expected to find it.

No discussion of 8th nerve tumors would be complete without a few notes concerning the other tumors to which the cerebellopontile angle is host. Among the most interesting are the meningiomas. Rather than eroding the petrous apex, meningiomas may produce hyperostosis in this region (9). Indeed, given a patient with clinical evidence of an 8th nerve tumor in whom eburnated bone is found in the region of the angle, one should strongly suspect a meningioma. We have seen one patient with startling osteosclerosis of the dorsum sellae and clivus in addition to increased density in the region of the porus, who proved to have a meningioma in the cerebellopontile angle (Fig. 24).

Slow growing cholesteatomas in this area

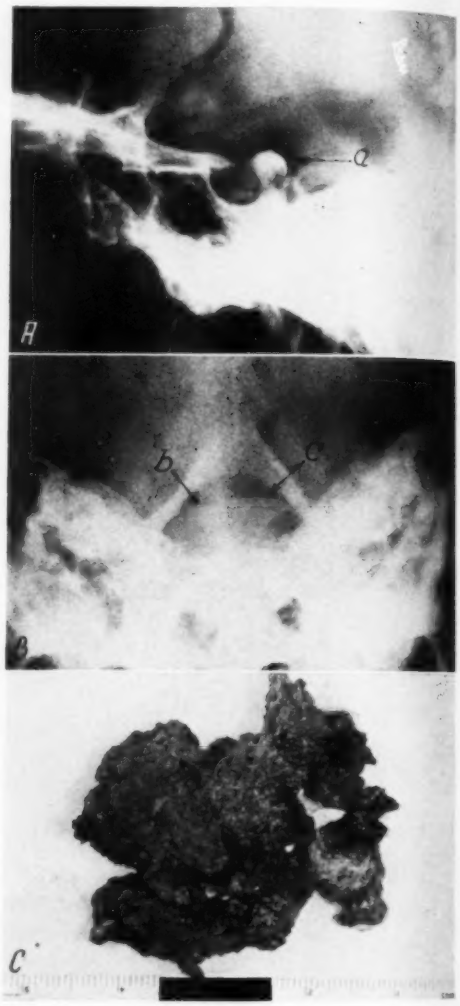

Fig. 24. Meningioma apparently springing from the region of the dorsum sellae, which produced clinical signs and symptoms of an 8th nerve tumor.
A. Lateral view. The hypophyseal fossa is normal in size and shape. The dorsum sellae is unusually dense, *a*, due to the hyperostosis commonly seen associated with meningiomas.
B. Occipital view. The dorsum sellae, *b* and *c*, is projected into the foramen magnum where the eburnated appearance is clearly defined.
C. Tumor tissue removed at operation.

may cause remarkable bone distortion of a degree not seen in 8th nerve tumors. In one of our patients the entire petrosal apex was displaced forward and the homolateral anterior clinoid pushed upward by the tumor (Fig. 25).

Nothing distinctive was noted in the cerebellopontile angle gliomas reviewed by us.

There are several clinical findings which may help differentiate true 8th nerve tumors from other growths in this area. Whereas acoustic tumors tend to follow a fairly definite order of symptoms, pontine growths are less prone to do so. Deafness, too, is by no means invariably present and complete.

Horrax and Buckley (25) called attention to the fact that papilledema is far less commonin true pontile growths than in 8th nerve tumors. Among 8 proved pontine lesions they found little or no choking of the optic disks in 7, even though there was clinical evidence of involvement of many cranial nerves. They pointed out that, were these 8th nerve tumors large enough to involve other cranial nerves, it would have been extremely unusual to find them unattended by increased intracranial pressure. These authors also observed that pontine tumors often cause bilateral symptoms early, in contradistinction to acoustic tumors.

SUMMARY

1. The 8th nerve lies in intimate relation with the 5th nerve, the 7th nerve, the nervus intermedius, and the anterior inferior cerebellar artery and vein, which course as a group through the cerebellopontine angle.

2. Because these structures adjoin each other, they are all usually affected by acoustic tumors.

3. The roentgen appearance of the pori acustici varies considerably in different heads and in both sides of the same head.

4. Histologically 8th nerve tumors fall into three broad classifications: neurinoma, von Recklinghausen's neurofibroma, and von Recklinghausen's neurofibromatosis associated with meningiomas.

5. Acoustic neurinomas are usually single lesions. When bilateral, they commonly belong to the neurofibroma group, which is often familial.

6. The clinical findings in acoustic tumors follow a somewhat characteristic progressive pattern. Early they are limited to the 5th, 7th, and 8th nerves. As the

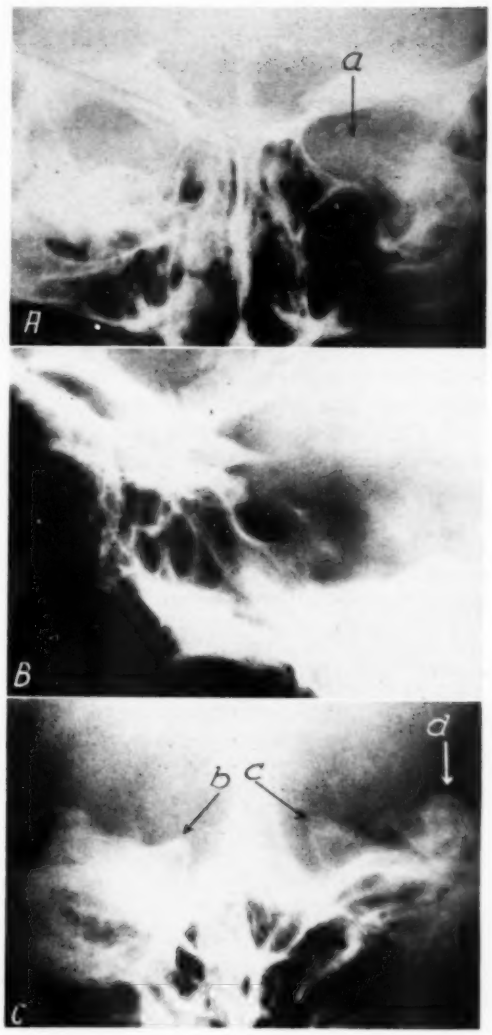

Fig. 25. Patient presented an 8th nerve syndrome on the left side, which proved to be due to a huge cholesteatoma.

A. Postero-anterior view. The apical portion of the left petrous pyramid is considerably eroded. The wing of the sphenoid, *a*, on the left side is also considerably demineralized.

B. Lateral view. Whereas the hypophyseal fossa is normal in size and shape, there is considerable atrophy of the dorsum sellae.

C. Postero-anterior projection with the head flexed upon the neck. The anterior clinoid processes are seen at *b* and *c*. The left anterior clinoid, *c*, is considerably distorted compared to the normal appearing right anterior clinoid, *b*.

tumors grow, they encroach upon the pons and cerebellum, producing ataxia, dysmetria, and adiadochokinesia. Finally, increased intracranial pressure supervenes.

7. Roentgenographically these tumors are characterized by erosion of the internal auditory canal. They commonly reveal also the roentgen manifestations of increased intracranial pressure.

8. Approximately 16 per cent of this series of 70 tumors revealed little or no evidence of erosion in the region of the internal auditory canal. Of these, many showed hypophyseal changes in the lateral view indicative of an intracranial lesion.

9. The evidence indicates that approximately 80 per cent of all 8th nerve tumors should be localized by roentgen methods.

NOTE: We wish again to thank Dr. Francis C. Grant, Dr. Robert A. Groff, and Dr. Michael Scott for allowing us free access to their records. Without the help of Dr. Grant's record librarian, Miss Anne Barnhart, our job would have been much more difficult. Dr. Edward Chamberlain and Dr. Arthur Finkelstein placed their film files at our disposal, and for this we are thankful. We are also indebted to Dr. Frederick H. Lewey and Dr. Eugene B. Spitz, who reviewed many histologic sections and advised us on the classification of questionable 8th nerve tumors. The illustrations were prepared by Mr. Reuben Goldberg.

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BIBLIOGRAPHY

1. ADELSTEIN, L. J., AND ANDERSON, F. M.: Tumor of Acoustic Nerve Within Petrous Bone; Operative Removal. Arch. Neurol. & Psychiat. **51:** 268–270, March 1944.

2. BAGER, C. C.: Differential Diagnosis between Acoustic Neurinoma and Meningioma of the Posterior Face of the Petrous-Bone. Acta psychiat. et neurol. **19:** 23–31, 1944.

3. BRAIN, W. R.: Diseases of the Nervous System. New York, Oxford University Press, 1947.

4. BRUNNER, H.: Brain Tumors and the Ear. Tr. Am. Acad. Ophth. **40:** 59–78, 1935.

5. CAMP, J. D., AND CILLEY, E. I. L.: Significance of Asymmetry of Pori Acustici as an Aid in Diagnosis of Eighth Nerve Tumors. Am. J. Roentgenol. **41**:713–718, May 1939.

6. CARR, G. L.: Roentgen Ray Findings in the Skull in Cases of Brain Tumors, with Special Reference to the Porus Acusticus. Am. J. Roentgenol. **4:** 405–410, 1917.

7. CHAMBERLAIN, W. E.: Personal communication.

8. Cabot case No. 24032: Acoustic Neuroma (Neurofibroma). New England J. Med. **218:** 127–129, Jan. 20, 1938.

9. COHEN, I.: Meningioma at Internal Auditory Meatus. J. Mt. Sinai Hosp. **10:** 206–207, May-June 1943.

10. CRAIG, W. M., AND STEENROD, E. J.: Bilateral Acoustic Neurofibromas. Arch. Otolaryng. **28:** 404–411, September 1938.

11. Cunningham's Text-book of Anatomy. New York, William Wood & Co., 1918.

12. CUSHING, H.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle. Philadelphia, Penna., W. B. Saunders Co., 1917.

13. CUSHING, H., AND EISENHARDT, L.: Meningiomas. Springfield, Ill., Charles C Thomas, 1938.

14. EBENIUS, B.: Results of Examination of the Petrous Bone in Auditory Nerve Tumors. Acta radiol. **15:** 284–290, 1934.

15. FOWLER, E. P., JR.: Acoustic Tumors within the Internal Auditory Meatus. Laryngoscope **46:** 616–627, August 1936.

16. FRIEDMAN, E.D : Atypical Acoustic Neuromas. J. Mt. Sinai Hosp. **9:** 435–445, November–December 1942.

17. GARDNER, W. J., AND FRAZIER, C. H.: Bilateral Acoustic Neurofibromas. Arch. Neurol. & Psychiat. **23:** 266–302, February 1930.

18. GARDNER, W. J.: Acoustic Tumors. S. Clin. North America **19:** 1225–1233, October 1939.

19. GARDNER, W. J., AND TURNER, O. A.: Multiple Intracranial Tumors: Discussion of Relation of Meningeal to Acoustic Tumors and Report of Case. J. A. M. A. **113:** 111–113, July 8, 1939.

20. GONZÁLEZ REVILLA, A.: Neurinomas of Cerebellopontine Recess; Clinical Study of 160 Cases Including Operative Mortality and End Results. Bull. Johns Hopkins Hosp. **80:** 254–296, May 1947.

21. GRANT, F. C., AND SPITZ, E.: Experiences with the Acoustic Neurinomas; Fifty Consecutive Cases. To be published.

22. HARDY, M., AND CROWE, S. J.: Early Asymptomatic Acoustic Tumor. Arch. Surg. **32:** 292–301, February 1936.

23. HENSCHEN, F.: Die Akustikustumoren, eine neue Gruppe radiographisch darstellbarer Hirtumoren. Fortschr. a. d. Geb. d. Röntgenstrahlen **18:** 207–216, 1911–12.

24. HENSCHEN, F.: Zur Histologie und Pathogenese der Kleinhirnbrückenwinkeltumoren. Arch. Psychiat. **56:** 20–122, 1915.

25. HORRAX, G., AND BUCKLEY, R. C.: Clinical Study of the Differentiation of Certain Pontile Tumors from Acoustic Tumors. Arch. Neurol. & Psychiat. **24:** 1217–1230, December 1930.

26. LYSHOLM, E.: Contribution to the Technique of Projection in Roentgenological Examinations of Pars Petrosa. Acta radiol. **9:** 54–64, 1928.

27. NIELSEN, A.: Acoustic Tumors, with Special Reference to End-Results and Sparing of Facial Nerve. Ann. Surg. **115:** 849–863, May 1942.

28. OLSEN, A., AND HORRAX, G.: Symptomatology of Acoustic Tumors with Special Reference to Atypical Features. J. Neurosurg. **1:** 371–378, November 1944.

29. PANCOAST, H. K., PENDERGRASS, E. P., AND SCHAEFFER, J. P.: The Head and Neck in Roentgen Diagnosis. Springfield, Ill., Charles C Thomas, 1940.

30. REEVES, D. L.: Bilateral Acoustic Neurofibromas; Report of Case in 16 Year Old Patient with Operative Removal and Autopsy Examination. Bull. Los Angeles Neurol. Soc. **6:** 91–103, September 1941.

31. SANTE, L. R.: Manual of Roentgenological Technique. Ann Arbor, Mich., Edwards Brothers, Inc., 1942.

32. SCHÜLLER, A.: Roentgen Diagnosis of Diseases of the Head. St. Louis, C. V. Mosby Co., 1918.

33. SCOTT, P.: Auditory Nerve Tumours. J. Laryng. & Otol. **53:** 772–781, 783–785, December 1938.

34. STIBBE, E. P.: Surgical Anatomy of Subtentorial Angle with Special Reference to Acoustic and Trigeminal Nerves. (Hunterian Lecture, Abridged.) Lancet 1: 859–862, April 15, 1939.

35. SVIEN, H. J., AND LOVE, J. G.: Cysts of Cerebellopontine Angle Simulating and Masking Acoustic Neurinoma: Report of 2 Cases. Proc. Staff Meet., Mayo Clinic. 17: 385–390, June 24, 1942.

36. TOWNE, E. B.: Erosion of the Petrous Bone by Acoustic Nerve Tumor; Demonstration by Roentgen Ray. Arch. Otolaryngology 4: 515–519, December, 1926.

SUMARIO

Tumores del Octavo Par: Manifestaciones Roentgenológicas

En este estudio de una serie de setenta tumores del VIII par para los cuales había radiografías, tratóse de determinar el papel del radiólogo en el diagnóstico de esas lesiones.

El VIII par hállase en íntima relación con los nervios del V y el VII pares y el intermediario y la arteria y la vena cerebelosas inferiores anteriores, que atraviesan en grupo el ángulo cerebelopontil. Por estar adyacentes estos tejidos, suelen afectarlos todos los tumores auditivos. Los hallazgos clínicos confórmanse a un patrón evolutivo algo típico. Al principio, limítanse al V, VII y VIII pares, pero al desarrollarse los tumores e invadir el puente de Varolio y el cerebelo, producen ataxia, dismetría y adiadococinesia, hasta que, por fin, sobreviene hipertensión intracraneal.

Los neurinomas acústicos, que constituyen 90 por ciento de todos los tumores del VIII par, suelen ser lesiones aisladas. Los tumores bilaterales corresponden por lo general al grupo neurofibromatoso, que es frecuentemente familiar.

Radiográficamente, caracterízanse estos neuromas en forma predominante por la erosión del conducto auditivo interno. Los AA. han empleado la siguiente clasificación arbitraria: (1) casos sin anomalías roentgenológicas en las petrosas, aunque suele haber signos de hipertensión intracraneana, (2) casos en que el único hallazgo roentgenológico es leve desosificación del conducto auditivo interno, (3) casos con ensanche, pero sin acortamiento, del conducto auditivo interno; (4) casos tanto con ensanche como acortamiento del conducto, que a menudo es infundibuliforme; (5) casos en que el conducto se halla totalmente destruído y en que las pirámides petrosas son todavía observables, aunque muy desmineralizadas; (6) casos con completa destrucción del conducto auditivo interno y del extremo del petroso.

Aproximadamente 16 por ciento de los casos de los AA. correspondieron a los Grupos 1 y 2, revelando pocos signos de erosión del conducto auditivo interno, pero en muchos de ellos, las vistas laterales mostraron alteraciones hipofisarias indicativas de lesión intracraneal.

A base de los datos así obtenidos, dedúcese que aproximadamente 80 por ciento de todos los tumores del VIII par deben ser localizados con técnicas roentgenológicas.

Roentgenologic Observations Concerning Erosion of the Sella Turcica[1]

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MANY OBSERVATIONS have been made in the past which indicate that the normal sella turcica varies widely in size and shape (1–3). So great is this variation that recognition of disease based on alterations of size alone is frequently difficult and even misleading. Rasmussen (4) has shown, also, that there is no constant relation between the size of the sella and the size of the pituitary gland. This further decreases the value of measurements of the sella alone as an index of pituitary disease. My own experience, over a period of many years, indicates that in the past the average roentgenologist has paid too much attention to variations in the dimensions of the sella turcica and has underestimated the significance of certain basic changes in structure which will indicate the presence of disease long before measurements alone have any value. These basic changes—erosion, decalcification and destruction of bone—are intimately related to one another. They may express themselves in focal or generalized changes in contour of the sella or its related processes, depending on the type and location of the lesion.

Erosion of the sella, or any of its related processes, is due to pressure from a contiguous mass, abnormal pulsation, or generalized pressure reflected from a more remote lesion. This focal or generalized pressure produces a loss of substance of the cortex of the exposed surface of the sella or contiguous processes. A certain amount of localized decalcification accompanies the pressure erosion, the degree being directly related to the site and severity of the associated pressure. If the pressure is of a low degree and persistent, it may be reflected only in a change in contour of a single process or of the entire sella with little evidence of decalcification, since in such cases there is a natural reparative

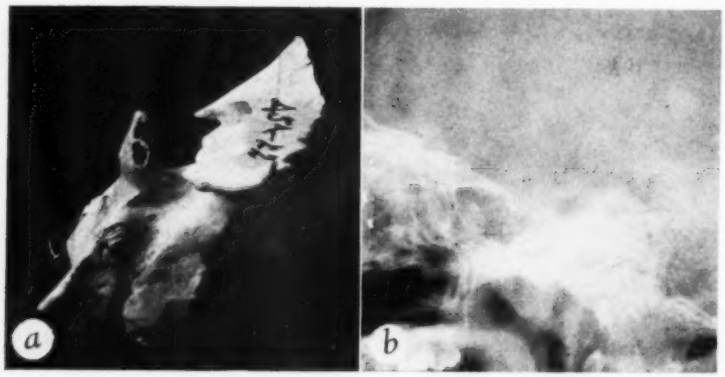

Fig. 1. *a*. Necropsy specimen revealing erosion of the posterior clinoid processes and floor of sella. Death occurred thirty-seven days following a bullet wound of the brain and nine days following clinical evidence of brain abscess and increased intracranial pressure. *b*. Lateral roentgenogram of skull made at necropsy. The indistinct fuzzy contour of the floor of the sella is evidence of the erosion revealed in Figure 1*a*.

[1] From the Section on Roentgenology, Mayo Clinic, Rochester, Minn. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

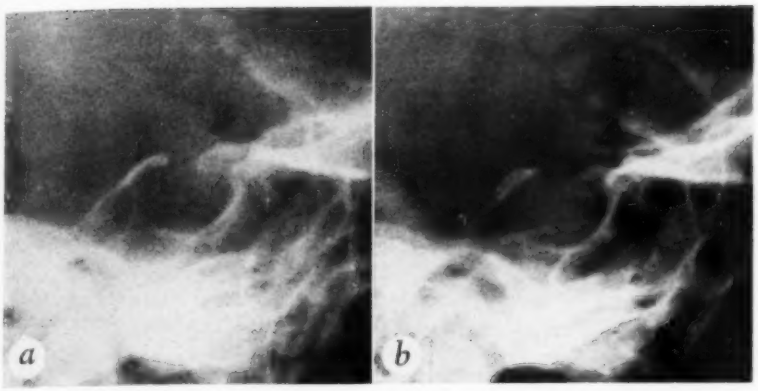

Fig. 2. Stereoscopic lateral views of the sella, indicating the value of shifting the x-ray tube along the coronal plane. *a*. The images of the anterior clinoid processes, which are superimposed, appear normal. *b*. After shifting tube, images of anterior clinoid processes are separated; focal erosion and foreshortening of right anterior clinoid due to pressure from a contiguous aneurysm of the right internal carotid artery are evident. Absence of shadow of the cortex of the bone at the site of erosion may be noted.

recalcification at the site of the erosion. On the other hand, if the pressure is acute, severe, and progressive, erosion occurs rapidly without opportunity for repair by recalcification, and the picture is soon one of complete loss of bone substance, either focal or generalized.

Classical examples of true decalcification of bone are seen in severe infections of the sphenoid sinus and, in varying degrees, in the presence of contiguous meningiomas. In the former, the decalcification results from the hyperemia of the walls of the sphenoid sinus which accompanies the infection, and in the latter both from hyperemia due to increased vascularity at the site of the tumor and from actual pressure by tumor cells which grow into the haversian canals of the adjacent bone.

Actual invasive destruction of the sella or its related processes occurs with malignant tumors which may originate in the bony structures or may involve them secondarily by extension from a contiguous soft-tissue mass. Such changes, because of their location and extent, are usually not difficult to distinguish from erosion incident to benign lesions. Extensive pressure erosion of the sella resulting from large pituitary tumors or generalized increased intracranial pressure may be imitated by a malignant lesion of the sphenoid bone, such as chordoma, myeloma, or carcinoma.

Fig. 3. Lateral view of skull revealing focal erosion and foreshortening of the right anterior clinoid process due to pressure from a contiguous calcified aneurysm of the right internal carotid artery. The changes are similar to those shown in Figure 2*b*, but the presence of calcium in the wall of the aneurysm contiguous to the eroded clinoid process facilitates an understanding of the mechanics of the erosion in such cases.

In any discussion relating to erosion, decalcification, or destruction of the sella, definite evidence indicating the relation of such changes to the duration of the primary disease would be of great value. A

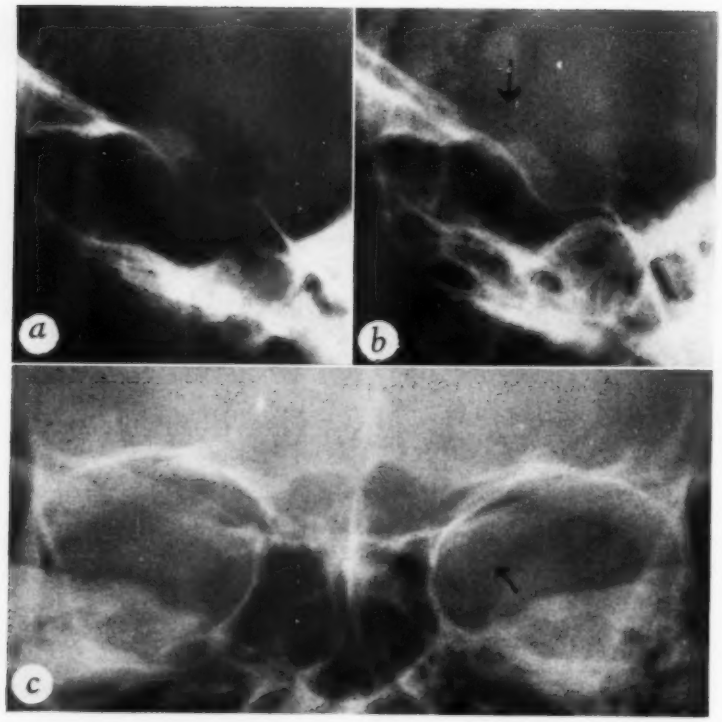

Fig. 4. Stereoscopic lateral roentgenograms of the skull. *a*. Images of the anterior clinoid processes are superimposed and therefore appear normal. *b*. Images of anterior clinoid processes have been separated by shift of tube along coronal plane; diffuse decalcification of the right anterior clinoid process is shown. *c*. Postero-anterior view, revealing diffuse decalcification of right anterior clinoid process due to meningioma of the right sphenoidal ridge.

number of years ago, while working on my thesis, I had the opportunity of studying the sella at necropsy in a case in which the duration of the primary disease was known. So far as I know, this is the only such case reported in the literature (1). The patient, a boy thirteen years of age, was accidentally shot in the head by a 22-caliber rifle and died thirty-seven days later as the result of a brain abscess. The necropsy specimen, shown in Figure 1, revealed focal erosion of the posterior clinoid processes and erosion of the floor of the sella. How long did it take to produce this erosion? Clinical signs of increased intracranial pressure did not develop until nine days before death. I think from this evidence that one can conservatively say that, in the presence of increased intracranial pressure, erosion or decalcification of the sella may occur within one month after the inception of abnormal pressure. The ability of the roentgenograms to reveal this change is dependent on the type of sella and whether or not it is undermined by a well pneumatized sphenoid sinus. A roentgenogram made after death in the case just cited (Fig. 1*b*) revealed early fuzziness of the contour of the floor of the sella, indicating decalcification and erosion secondary to the increased intracranial pressure. The changes were evident so early in this case only because the sella was partially undermined by a pneumatized sphenoid sinus which afforded a good contrasting density. If the sphenoid below the floor of the sella is not pneumatized—and it frequently is not in children before the age of twelve years—erosion of the floor may not be recognized

roentgenographically until more extensive changes have occurred.

Roentgenographic technic plays an important part in contributing to the early recognition of changes in the sella and its related processes. Stereoscopic roentgenograms are necessary, but these, to be of greatest value in this region, must be made with the tube shifted along the coronal plane. For the first roentgenogram, the tube should be centered to produce an exact lateral view of the sella. This is important in the portrayal of early changes in the floor. The shift of the tube for the second roentgenogram will separate the images of the clinoid processes and the edges of the sella, a very important maneuver since it facilitates the recognition of focal changes which may involve only one process or side of the sella. This is well shown in Figure 2*a* and *b*, in which the focal erosion and foreshortening of the right anterior clinoid process was due to an aneurysm of the internal carotid artery. When such changes are recognized, the roentgenologist can be very precise and exact in the localization of the lesion. The mechanics of this erosion are well shown in Figure 3, in a case in which the walls of an aneurysm of the internal carotid artery were partially calcified. Foreshortening of the contiguous anterior clinoid process due to pressure erosion may be seen. At the point of contact with the wall of the aneurysm, the cortex of the anterior clinoid process has disappeared as a result of pressure and decalcification. Normally the clinoid processes are outlined by a shadow of thin cortical bone. Dehiscence in this shadow is the first indication of contiguous pressure.

The images of the anterior clinoid processes are superimposed in Figure 4*a*, and no abnormality is evident. In Figure 4*b*, however, which is the matching stereoscopic roentgenogram, the images of the anterior clinoid processes are separated, and it is obvious that there is a great difference in density between one of these and the other. This same difference is also apparent in Figure 4*c*, which is a postero-anterior view.

Fig. 5. The postero-anterior 107-degree projection of skull (Granger position) revealing focal erosion and defect in continuity of the so-called G line as the result of a contiguous meningioma of the left optic nerve sheath.

In this case the tumor cells from an adjacent meningioma have invaded the bone and, with the associated increased vascularity, have produced diffuse decalcification which results in a diffuse, hazy image, in contrast with the focal type of pressure erosion shown in Figures 2 and 3. The roentgenologist, therefore, if he has good roentgenograms and has had reasonable experience, may be able to predict the type of contiguous lesion from the observation of such changes.

The postero-anterior 107-degree angle view, originated by Granger (5), for depicting disease of the sphenoid sinus, is valuable also for revealing and lateralizing focal changes resulting from lesions about the sella, tuberculum, or optic nerves. Local changes in bone which consist of defects in continuity (Fig. 5) or abnormal thickening (Fig. 6*a* and *b*) of the so-called G line may be more clearly defined in roentgenograms obtained in this position than in conventional lateral or postero-anterior views. The osteomatous changes resulting from meningiomas originating about the tuberculum sellae and posterior portion of the olfactory groove are especially well shown in the views taken in the Granger position. Such tumors, as they enlarge, frequently produce erosive changes in the floor of the

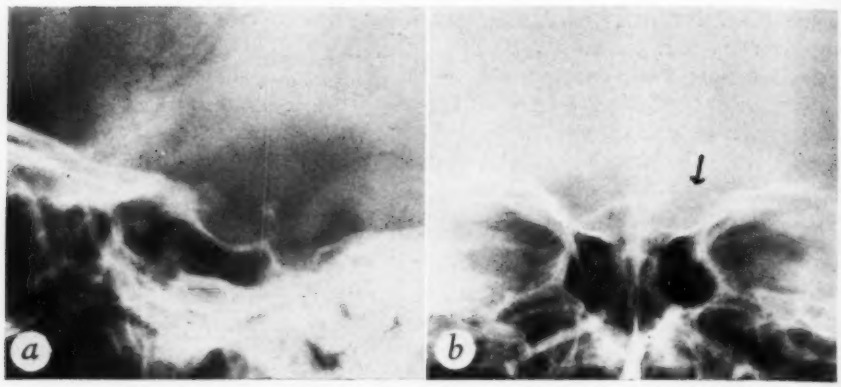

Fig. 6. Meningioma arising from tuberculum sellae. *a*. Lateral view of sella, revealing osteoma at site of origin of tumor and early pressure erosion of floor of sella. *b*. Postero-anterior view of skull, revealing osteomatous thickening of so-called G line at site of tumor.

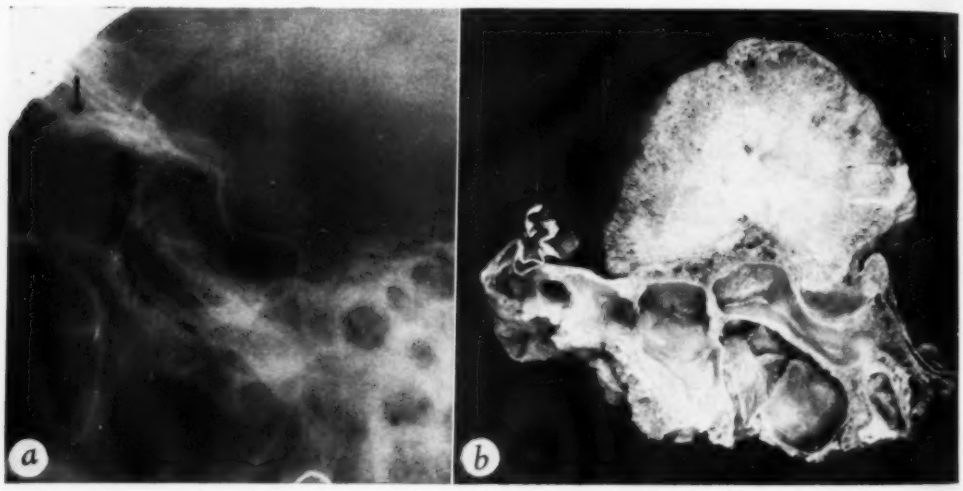

Fig. 7. *a*. Lateral view of sella, revealing osteomatous thickening of olfactory groove and tuberculum sellae resulting from contiguous meningioma. Note the decalcification of the floor of the sella and double contour due to pressure erosion. *b*. Necropsy specimen cut in sagittal plane through tumor and sella. Note erosion and osteomatous changes at point of attachment of tumor. The tumor has extended partially into the sella turcica and produced pressure erosion of the floor of the sella.

sella and posterior clinoid processes (Fig. 7*a*), the mechanics of which are well shown in Figure 7*b*, which is a sagittal section through such a tumor and the sella.

Erosion of the posterior clinoid processes may be the result of (1) direct pressure from a contiguous tumor, most commonly a partially calcified craniopharyngioma (Fig. 8), (2) pressure from a dilated third ventricle (Fig. 9), and (3) mass lesions of the brain without hydrocephalus which produce indirect pressure on the clinoid processes and sella (Fig. 10).

In the past it was not uncommon for roentgenologists to accept the presence of decalcification and circular expansion of the sella as evidence of a primary intrasellar tumor (Fig. 11). Experience has shown, however, that these changes may be produced, also, by tumors located elsewhere in the cranial cavity (Figs. 12 and 13). In such cases a displacement of the shadow of

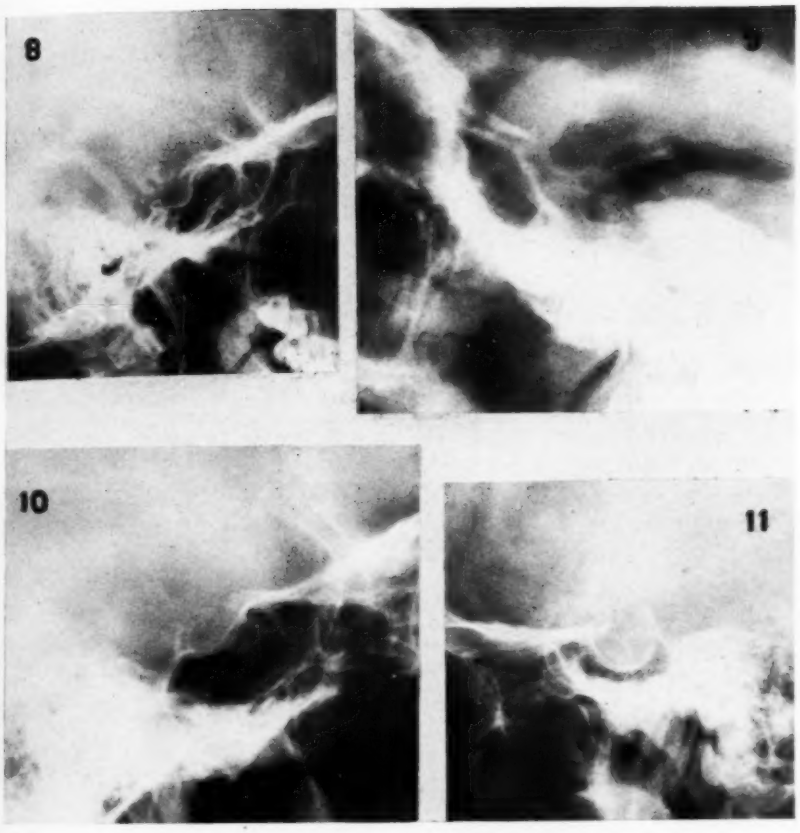

Fig. 8. Calcified craniopharyngioma producing focal erosion of the posterior clinoid processes and dorsum sellae.
Fig. 9. Ventriculogram revealing dilated third ventricle with erosion of posterior clinoid processes and sella as a result of hydrocephalus due to a cerebellar tumor.
Fig. 10. Astrocytoma of left temporal lobe producing focal erosion of posterior clinoid processes by mass pressure without hydrocephalus.
Fig. 11. Circular expansion and decalcification of the sella produced by primary intrasellar pituitary tumor.

a calcified pineal gland or evidence of generalized increased intracranial pressure will provide a clue to the extrasellar location of the lesion. Such evidence may, of course, be lacking, and it is therefore important to remember that whenever the roentgenogram suggests an intrasellar tumor and the clinical evidence does not agree, additional methods of localizing the lesion, such as encephalography, ventriculography, or arteriography, must be considered.

The capacity of bone to recalcify after pressure on it has been relieved or an invading tumor has been destroyed by radiation is well illustrated by the sella turcica and its related processes. Roentgenologic evidence of such recalcification is important evidence of a favorable response to treatment (Fig. 14*a* and *b*). This phenomenon is particularly well shown in radiosensitive malignant tumors of the sphenoid which invade and destroy the sella without actual enlargement of the pituitary fossa (Fig. 15*a* and *b*). Since the sella is not expanded, measurements are of no diagnostic value, and the significant roentgenologic signs are those of destruction and subsequent repair of bone.

When the sella has been decalcified and

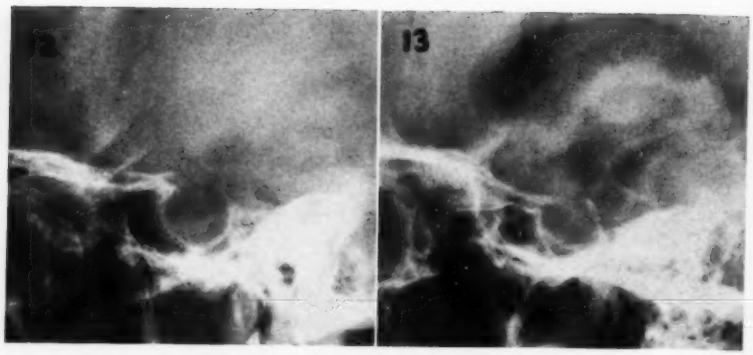

Fig. 12. Circular expansion and decalcification of the sella produced by right temporal lobe tumor. The changes simulate those of intrasellar tumor.
Fig. 13. Circular expansion and decalcification of the sella produced by indirect pressure and hydrocephalus incident to a neurofibroma of the right acoustic nerve. Air in ventricular system. Changes simulate those of intrasellar tumor.

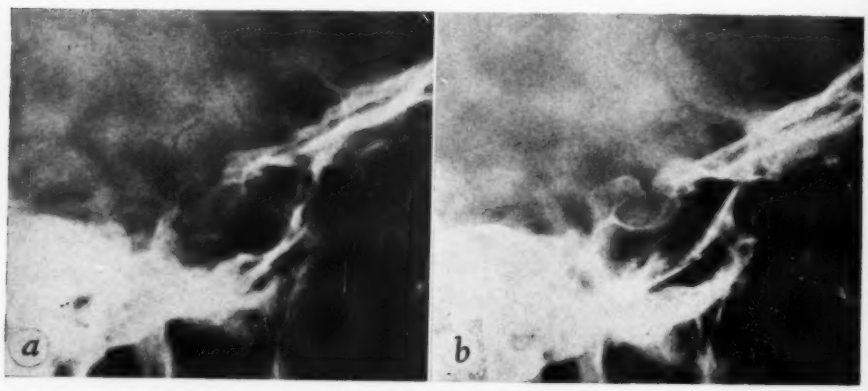

Fig. 14. *a*. Secondary erosion and decalcification of the sella and posterior clinoid processes resulting from pressure incident to a glioblastoma of the pons. *b*. Recalcification of sella and restoration of normal contour four months after roentgen treatment to tumor.

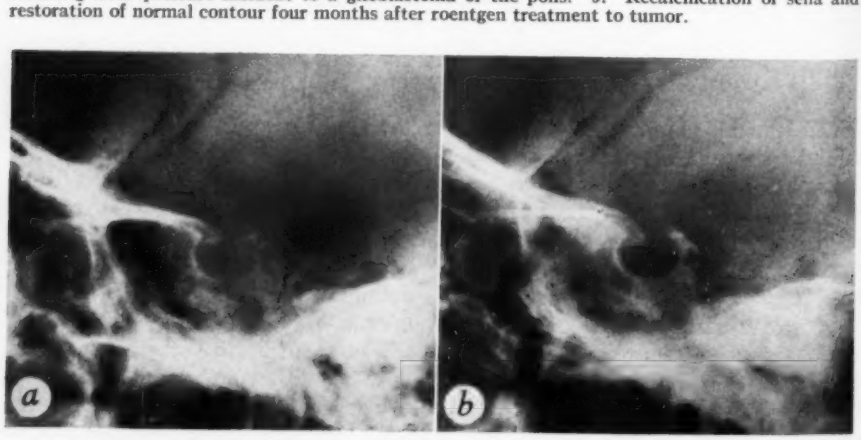

Fig. 15. *a*. Invasion of sphenoid bone and destruction of posterior portion of sella caused by Grade 4 epithelioma of sphenoid sinus. *b*. Appearance of sella five months after radium treatment. Note recalcification and restoration of contour of sella.

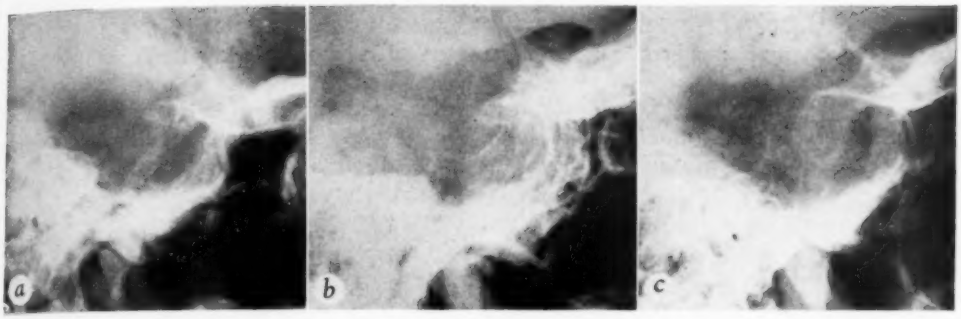

Fig. 16. Chromophobe adenoma of pituitary. *a*. Decalcification and enlargement of sella before surgical removal of tumor in 1935. *b*. Recalcification of sella six years after operation and roentgen therapy in 1941. There is no clinical evidence of activity of tumor. *c*. Extensive decalcification and further enlargement of sella due to recurrence of tumor in 1946.

enlarged by pressure from an intrasellar or extrasellar lesion and the pressure has been subsequently removed by one means or another, the margins of the sella will recalcify, but the pituitary fossa will maintain its abnormal size and shape (Fig. 16*a* and *b*). A roentgenogram will always reveal this evidence of antecedent disease, and it should not be interpreted as evidence of an active process. In this connection, serial roentgenographic studies made before, during, and subsequent to treatment are most important in the evaluation of the prognosis.

When an intracranial tumor recurs and the pressure phenomena of the original disease are re-enacted, the sella, which had recalcified following treatment of the lesion, will again undergo decalcification, and this important evidence of recurrence will be readily apparent if previous roentgenograms are available for comparison (Fig. 16*a*, *b*, and *c*). Here again it is the character and density of the bony margins of the sella that indicate disease, and not primarily any variation in size.

In summary, I would like to emphasize: (1) the importance of focal changes in the bony contour of the sella turcica and its related processes as an index of disease before any significant change in size of the sella occurs; (2) the value of decalcification and recalcification of the contour of the sella in following the course of intracranial disease; (3) the fact that the circular uniformly enlarged sella, long recognized as characteristic of a pituitary tumor, may be produced by tumors situated elsewhere within the cranial cavity; (4) the indications for supplementary methods of localization, such as ventriculography, encephalography, and arteriography, when the clinical and roentgenologic findings do not agree.

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REFERENCES

1. CAMP, J. D.: Normal and Pathologic Anatomy of the Sella Turcica as Revealed at Necropsy. Radiology **1:** 65–73, October 1923.

2. CAMP, J. D.: Normal and Pathologic Anatomy of the Sella Turcica as Revealed by Roentgenograms. Am. J. Roentgenol. **12:** 143–156, August 1924.

3. GORDON, M. B., AND BELL, A. L. L.: A Roentgenographic Study of the Sella Turcica in Normal Children. New York State J. Med. **22:** 54–58, February 1922.

4. RASMUSSEN, A. T.: A Quantitative Study of the Human Hypophysis Cerebri, or Pituitary Body. Endocrinology **8:** 509–524, July 1924.

5. GRANGER, A.: Positive Identification of Sphenoid and Ethmoid Sinuses. J. A. M. A. **81:** 1336–1340, Oct. 20, 1923.

(*For Spanish summary, see following page*)

SUMARIO

Observaciones Roentgenológicas acerca de la Erosión de la Silla Turca

Parece que, en el pasado, exageraron la atención prestada al tamaño de la silla turca como índice de afección intracraneal y menospreciaron ciertas alteraciones histológicas básicas que la afectan, a saber: erosión, descalcificación y destrucción ósea. Esas alteraciones básicas pueden traducirse por alteraciones focales o generalizadas del contorno de la silla o de sus apófisis, conforme al tipo y la localización de las lesiones.

La erosión de la silla o de sus apófisis, se debe a compresión por alguna masa contigua, pulsación anormal o presión generalizada reflejada de una lesión más remota, yendo acompañada de cierta proporción de descalcificación localizada. Obsérvanse ejemplos clásicos de verdadera descalcificación en las infecciones esfenoidales graves y, en mayor o menor grado, en presencia de meningiomas contiguos. Destrucción invasora real de la silla o de sus apófisis tiene lugar en los tumores malignos que pueden tener su origen en los tejidos óseos o invadir éstos secundariamente por difusión de una tumefacción de los tejidos blandos adyacentes.

Se hace hincapié en particular en los siguientes puntos: (1) importancia de las alteraciones focales del contorno óseo de la silla y de sus apófisis como signo de enfermedad antes de que ocurra la menor alteración significativa del tamaño de la silla; (2) valor de la descalcificación y la recalcificación del contorno de la silla para seguir la evolución de una afección intracraneana; (3) el hecho de que los tumores de otras partes de la cavidad craneal pueden producir la uniforme hipertrofia circular de la silla, reconocida de viejo como típica de tumor hipofisario; (4) importancia de los métodos complementarios de examen, tales como ventriculografía, encefalografía y arteriografía, cuando no concuerdan los hallazgos clínicos y radiográficos.

The Roentgen Diagnosis of Meningiomas of the Sphenoidal Ridge[1]

EUGENE P. PENDERGRASS, M. D., JOHN W. HOPE, M. D.,[2] and CHARLES R. PERRYMAN, M. D.

MENINGIOMAS, which constitute about 15 per cent of all brain tumors, are among the few intracranial neoplasms which lend themselves to successful surgical removal. For this reason, any diagnostic assistance the roentgenologist can offer is of practical value. The roentgenogram alone will not always establish the diagnosis of meningioma. It is true that the roentgen findings are often so typical that no other diagnosis would be logical, but enough atypical cases are constantly occurring to bring out the fact that correct interpretation of the roentgenogram may depend on knowledge of the tumor itself, the history of the patient, and careful physical and neurologic examinations. Even with all this information at hand, the diagnosis is not always easy. There is need for close cooperation between the neurosurgeon, the neurologist, and the roentgenologist.

HISTOGENESIS

That the histogenesis of meningiomas has long been obscure is obvious from the number of terms by which they have been known. Chapter I of Cushing and Eisenhardt's monograph on meningiomas (1) is devoted to the historical background of this tumor, and in this source the following terms appear:

fungus durae matris (Louis, 1774)
tumeurs cancéreuses des méninges (Cruveilhier 1835)
sarcoma (Virchow, 1847)
tumeurs fibro-plastiques intra-crâniennes (Lebert, 1849)
psammoma (Virchow, 1850)
epithelioma (Meyer, 1859)
endothelioma (Golgi, 1869)
arachnoid fibroblastoma (Mallory, 1920)
meningioma (Cushing, 1922)
meningeal fibroblastoma (Penfield, 1927).

At first these tumors were thought to arise from the dura mater. Virchow called them sarcomas, but later adopted the term psammoma or sand tumor, because of the number of small concretions found within them. As they sometimes resembled epithelial cancer, the term epithelioma came into use. Later, epithelioma was replaced by endothelioma, since the term endothelium was employed to designate membranes of mesodermal origin lining body cavities, and the meshwork of the meninges was regarded as such a membrane. Mallory called the tumors arachnoid fibroblastomas because he thought they were made up of undifferentiated potential fibroblasts. Cushing arbitrarily coined the word meningioma as a nonspecific designation. The two terms fibroblastoma and meningioma are still used, but of the two, meningioma seems to be the more widely accepted.

The identity of these tumors would be more obvious if the membrane from which they arise were known. The arachnoid villi are continuations of the arachnoid mesh into the lateral walls of the great dural sinuses. The arachnoid cells at the tips of the villi, directly below the vascular endothelium, are usually several layers in thickness, so that cellular caps over the villi are formed (2). The cells making up these villous caps resemble those found in meningiomas, and it is because of this striking similarity that meningiomas are thought to arise from the arachnoid (1, 3, 4). Further substantiation of this origin is found in the fact that meningiomas most frequently occur along the course of the dural venous sinuses. As part of the normal aging process, these arachnoidal villi become greatly enlarged, thickened,

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[2] Trainee, National Cancer Institute.

and even calcified, and are then known as pacchionian granulations (3). The roentgenologist is well aware of the areas of rarefaction or of increased density appearing in the inner table of the skull at the vertex and in the frontal and parietal regions due to these pacchionian granulations (5).

Assuming that meningiomas arise from the arachnoid, their identity would be better understood if the histogenesis of the arachnoid were known. In 1926, Ariens Kappers (6), on the basis of histologic study, wrote as follows: "The most probable relation seems to be that the primitive leptomeninx is independent in its origin from that of the dural membrane, and that the real dura develops from the mesenchymatous blastema immediately surrounding the leptomeninx, *i. e.*, from the perimeningeal tissue but not from the endosteum of the vertebrae from which only the so-called external or periosteal lamella arises."

Later in 1926, Harvey and Burr (7) demonstrated by experimental transplants in *Amblystoma punctatum* that the primordium of the leptomeninx is derived from the ectodermal neural crest, and that of the pachymeninx from the mesenchyme. These experiments were confirmed in the chick and pig embryo.

In 1933 Harvey, Burr, and Van Campenhout (8) presented further experimental work in which they used chorioallantoic grafts of the nervous system of the chick. They showed that transplants of the neural tube with the neural crest possess a characteristic cellular membrane on the outer surface of the neural tube, whereas transplants without the neural crest showed a complete absence of this cellular layer. They also selectively stained the neural crest with Nile blue sulfate and found Nile blue-stained cells surrounding the unstained nervous system, with similarly unstained mesenchyme surrounding the whole. These experiments further confirmed their original thesis that in the development of the meninges the primordium of the leptomeninx is derived from the neural crest and that of the pachymeninx from the mesenchyme.

In this same year Leary and Edwards (9), in a study of subdural hemorrhages, noted the great differences between the functions and reactions of the arachnoid and the dura. These differences led them to take scrapings of the pericardial and peritoneal cavities and to compare the sheets of cells obtained with scrapings from the arachnoid and the dura. They concluded that the dura is lined with fibroblastic tissue and that the arachnoid is covered with cells which do not appear to be mesothelial, but are probably of ectodermal origin.

In 1935 Globus (10) presented another concept, namely, that all three of the meninges arise from a mesenchymal tissue which fills the space between the skin and the neuroectoderm; he referred to this as skeleto-neural intertissue. This mesenchymal tissue then splits into two parts, an internal and an external. The internal part becomes the pia and the arachnoid, and the external part the dura and the periosteum. Globus was of the opinion that meningiomas arise from this primitive mesenchymal tissue, and that, in this manner, they may acquire the character of any of the component layers of the meninges. He regarded them as compound tumors in which either the parenchymatous, epithelioid element, or the fibroblastic, stromal element may predominate. In 1944 Globus (11) still held to this concept.

Whether these tumors arise from neuroectoderm or from mesenchyme is still an unsettled question, which it is not the aim of this discussion to resolve. The matter has been presented solely to emphasize to the roentgenologist that the histogenesis of this tumor is still undetermined.

GROSS AND MICROSCOPIC PATHOLOGY

Grossly, the tumors are of two forms: massive or global and so-called *meningioma en plaque*. The massive or global form is not likely to produce severe hyperostosis of the overlying bone, but usually invagi-

TABLE I: THE NINE MAJOR HISTOLOGIC TYPES OF MENINGIOMA (CUSHING AND EISENHARDT)

Type I. Non-reticulin- or collagen-forming meningothelial tumors
- Var. 1. Cells evenly distributed in sheets
- Var. 2. Cells in alveolar arrangement
- Var. 3. Spindle cells in interlacing bundles
- Var. 4. Combination of variants 1, 2 and 3

Type II. Meningothelial tumors of whorl pattern with tendency to form reticulin or collagen
- Var. 1. Variously distributed spindle, round, and polyhedral cells
- Var. 2. Combination of Type I, var. 1 or 2, with whorls
- Var. 3. Uniform small whorls
- Var. 4. Compact psammoma bodies

Type III. Reticulin- or collagen-forming fibroblastic tumors of benign type
- Var. 1. Fibrils scanty
- Var. 2. Fibrils abundant
- Var. 3. Marked fibrosis

Type IV. Reticulin-forming angioblastic tumors
- Var. 1. Incompletely differentiated, with mitoses
- Var. 2. Transitional between meningothelial type and angioblastoma
- Var. 3. Angioblastoma (capillary or cellular)

Type V. Non-reticulin- or collagen-forming epithelioid tumors
- Var. 1. Cells in columnar arrangement
- Var. 2. Roussy, Cornil, and Oberling type

Type VI. Reticulin- or collagen-forming fibroblastic tumors of malignant type (sarcomatous meningiomas)
- Var. 1. Spindle-cell type
- Var. 2. Round-cell type

Type VII. Osteoblastic meningiomas

Type VIII. Chondroblastic meningiomas
- Var. 1. Chondroma
- Var. 2. Osteochondroma

Type IX. Lipoblastic meningiomas

nates the pia into the brain, and may become completely buried. The flat spreading carpet of tumor called *meningioma en plaque* is sessile, has a limited thickness, and grows along the plane of the meninges. It is this flat form which usually produces a severe degree of hyperostosis.

Cushing and Eisenhardt (1) have presented a comprehensive histological classification in which all meningiomas are sub-divided into nine major types with several variants under each of these types. These they have arranged in the order of their numerical frequency, with the most common type heading the list. Cushing and Eisenhardt stressed the point that over the course of years these tumors rarely, if ever, tend to alter in type. This classification is presented (Table I) because it has been adopted by the Department of Neuropathology, University of Pennsylvania.

It is appropriate that the question of the degree of malignancy be discussed at this point. Cushing and Eisenhardt (1) stated that the great majority of meningiomas are relatively benign lesions usually limited internally by the pia, and externally by the galea aponeurotica. In 1935 Globus (10) and in 1944 Globus, Levin, and Sheps (11) stated that in a number of cases meningiomas invaded the brain tissue by way of the pial blood vessels. In 1942 Turner, Craig, and Kernohan (12) concluded that about 10 per cent of the intracranial meningiomas of their series were microscopically malignant when judged by the type of cell, the architecture of the tissue, and the presence of giant-cell forms and mitotic figures. In 1944, Globus, Levin, and Sheps (11) concluded that a little less than 10 per cent of the tumors in their series were malignant. The malignant feature in all of these was local invasion rather than distant metastases. This local invasiveness seems to substantiate Globus' observation that meningiomas penetrate the pial barrier.

Meningiomas rarely give rise to distant metastases. Cushing and Eisenhardt (1) reported one case, and a case has been recorded by Globus, Levin, and Sheps (11). Haymaker (13), in 1948, reviewed the literature and found five examples of distant metastasis from a primary meningioma. In our series, Case 17 gave rise to pulmonary metastases.

This whole question of the degree of malignancy is largely academic, for over the years it has been demonstrated that postoperative results are usually good if a successful surgical removal of all the visible tumor has been performed.

BONY CHANGES PRODUCED BY MENINGIOMAS

Meningiomas are capable of causing erosion, absorption, hyperostosis, or eburnation of bone, either through actual infiltration of the haversian canals or by the reactive hyperemia they provoke (14). Cushing and Eisenhardt have shown these bony changes diagrammatically in Figure 10 (page 21) of their book on "Meningiomas" (1). The radiologist who is inter-

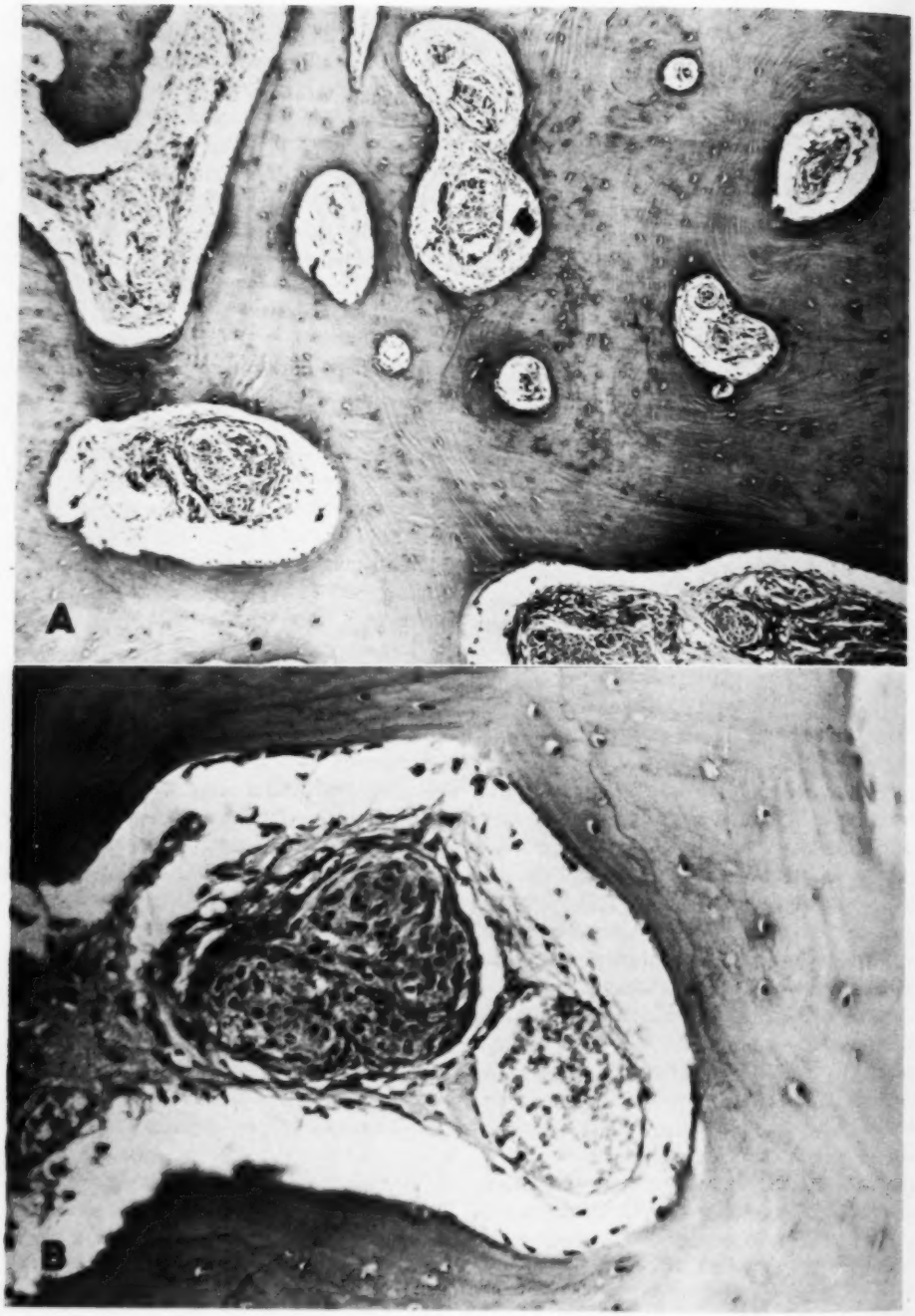

Fig. 1. Bone removed from the sphenoidal ridge which has been invaded by a meningioma Type II, variant 1. Low- and high-power magnifications. Hematoxylin and eosin. (Photomicrographs courtesy of Armed Forces Institute of Pathology. Accession number: 218225–1 and 218225–2.)

ested in this problem should familiarize himself with this diagram. These authors described eight varieties of bone involvement, which have been summarized in our Table II.

TABLE II: EIGHT VARIETIES OF BONE INVOLVEMENT PRODUCED BY MENINGIOMAS

1. Increased vascularity of bone.
2. Thinning of the bone by pressure absorption.
3. Enostosis projecting into the substance of the tumor with no visible thickening of the outer table.
4. Thickening of both tables producing a palpable and visible swelling on the scalp.
5. Dense eburnation of the outer table.
6. Hyperostosis with vertical striations.
7. Hyperostosis with an extracranial pancake of tumor lying between the hyperostosis and the galea.
8. Hyperostosis in which the central core of the hyperostosis is completely destroyed by the tumor.

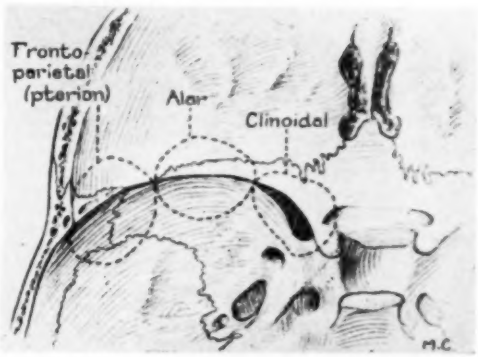

Fig. 2. Three divisions of the sphenoidal ridge that give rise to symptomatically distinguishable meningiomas. (From Cushing and Eisenhardt; Meningiomas: Their Classification, Regional Behaviour, Life History, and Surgical End Results, p. 298. Courtesy of Charles C Thomas.)

Sometimes the hyperostosis contains tumor cells, but sometimes it does not. This would seem to indicate that the bone thickening is probably due to the increased vascularity, with tumor invasion a secondary occurrence which may or may not take place (Fig. 1).

Cushing (15), in 1922, stated that there was a tendency for the meningioma cells to work through the vascular spaces in the dura and finally reach the canalicular spaces of the bone, but not to infiltrate, in the sense of malignant-cell infiltration. The cells then stimulate osteoblastic proliferation around their nests, giving the appearance of spicules.

Phemister (16) believes that the new bone is reactive in nature and is an integral part of the tumor. Kolodny (17) believes that the hyperostosis is the result of an early, slow, and progressive dilatation of the blood vessels in the cranial bones overlying the meningioma, and that infiltration (not penetration) by the tumor subsequently takes place and leads to bone destruction.

Rowbotham (18), discussing the circulation of meningiomas and its relation to the hyperostosis, states that the blood supply is out of proportion to the size of the tumor. The arterial supply from the dura, with secondary communications with the pial vessels, extends to the tumor capsule. The primitive vessels do not have any sympathetic vasoconstrictor control. There are sinusoids and abnormal communications between the arteries and veins which produce a bed of low endovascular resistance. Any excess of blood finds its way through the tumor, due to the absence of arteriolar and capillary resistance. There are important communications between the dural veins and those of the diploe in the meningeal grooves and in the many pits seen in the inner table of the skull. These channels dilate and are responsible for the changes seen in the roentgenograms.

Rowbotham (18) recognizes five types of bone change: (1) the diffuse tumorous hyperostoses of the vault; (2) the diffuse hyperostosis of the base; (3) osteoclastic hyperostosis; (4) nodal enostosis; (5) diffuse non-tumorous hyperostosis.

Not only are there changes in the adjacent bone, but sometimes calcification takes place within the tumor itself. The microscopic manifestations of this calcification are spoken of as psammoma bodies. Globus (10) believes that psammoma bodies are nothing but vascular buds ending blindly, thus blocking free circulation of the enclosed blood. The result is that the stagnant blood, as well as the enclosing vascular wall, undergoes degeneration, hyalinization, and calcification.

These changes, either within the tumor or

TABLE III: POINT OF ORIGIN OF 295 INTRACRANIAL MENINGIOMAS (CUSHING AND EISENHARDT)

I.	MENINGIOMAS OF THE VAULT (131 cases)		44.4%
	A. Parasagittal (65 cases)	22%	
	1. Non-hyperostosing (*global*) (51 cases)		
	2. Hyperostosing (*en plaque*) (14 cases)		
	B. Lateral (54 cases)	18.4%	
	1. Precoronal (5 cases)		
	2. Coronal (17 cases)		
	3. Postcoronal (7 cases)		
	4. Pararolandic (9 cases)		
	5. Parietal (12 cases)		
	6. Temporal (4 cases)		
	7. Occipital (0 case)		
	C. Peritorcular (12 cases)	4%	
II.	MENINGIOMAS OF THE BASE (110 cases)		37.3%
	A. Sphenoidal ridge (53 cases)	18%	
	1. Inner third (13 cases)		
	2. Middle third (11 cases)		
	3. Outer third (29 cases)		
	(a) *Global* (13 cases)		
	(b) *En plaque* (16 cases)		
	B. Olfactory groove (29 cases)	9.8%	
	C. Suprasellar (28 cases)	9.4%	
III.	POSTERIOR FOSSA MENINGIOMAS (23 cases)		7.8%
	A. Cerebellar (15 cases)	5.1%	
	B. Cerebellopontile angle (7 cases)	2.4%	
	C. Basilar groove (1 case)	0.3%	
IV.	MISCELLANEOUS (31 cases)		10.5%
	A. Infratemporal (8 cases)	2.7%	
	B. Tumors of the falx (7 cases)	2.4%	
	C. Without dural attachment (6 cases)	2.1%	
	D. Cavum mecklii (5 cases)	1.8%	
	E. Multiple (2 cases)	0.6%	
	F. Combined with neurinomas (2 cases)	0.6%	
	G. Intraorbital (1 case)	0.3%	

in the adjacent bone, produce the characteristic roentgen findings on which a diagnosis is so often made. Grant (19) reported a series of 149 intracranial meningiomas and stated that the diagnosis was suggested on the flat x-ray films in 81 of the cases. In another 20 cases the presence of a mass was definite on the basis of the pineal shift. In the 48 cases which were originally reported as negative, restudy of the roentgenograms, after the position of the tumor was known, showed suggestively positive findings in 14. When ventriculography, encephalography, or arteriography is resorted to, the percentage of positive diagnoses is still higher.

POSITION OF THE TUMOR

Meningiomas tend to grow in a definite regional distribution. They can be broadly divided into tumors of the vault and tumors of the base. Cushing and Eisenhardt (1) have gone into great detail in describing the exact position and point of origin of each intracranial meningioma in their series of 295. Table III is a summary of their 295 cases arranged in the relative frequency with which they appear in certain locations, expressed in percentages. From this table one can see at a glance where meningiomas tend to occur most frequently. A knowledge of where these tumors arise should aid the roentgenologist in arriving at a diagnosis, and it is for this reason that Table III has been prepared and included.

SPHENOIDAL RIDGE MENINGIOMAS

In this presentation meningiomas of the sphenoidal ridge only will be discussed.

From Jan. 1, 1930, to Jan. 1, 1949, a total of 1,802 brain tumors have been verified in the Neurosurgical Clinics of the University and Postgraduate Hospitals of the University of Pennsylvania. Of this number, 288 (15.9 per cent) have been meningiomas, and of this group, 41 (14.5 per cent) were meningiomas of the sphenoidal ridge. This agrees quite closely with Cushing and Eisenhardt's 18 per cent shown in Table III.

The sphenoparietal sinus runs along the

sphenoidal ridge, and meningiomas of this region probably arise from the cells capping the arachnoid villi which project into this sinus. Cushing and Eisenhardt (1) divide meningiomas arising from the sphenoidal ridge into three groups: (1) deep, inner or clinoidal; (2) middle or alar; (3) outer or pterional. The pterional group is further subdivided into *global*—non-hyperostosing lesions—and *en plaque*—hyperostosing lesions (Fig. 2).

Grant (19), who operated on the majority of the 41 cases in our series, does not believe that it is always possible to determine the tumor's exact point of origin even with careful examination of the preoperative roentgenograms and painstaking perusal of the operative notes. Because of this, we have not attempted to divide our 41 cases into subgroups, but have simply presented them in the chronological order of their admission to the Hospital (pp. 690–706).

Of the 41 patients in our series, 28 were females and 13 were males. One of the females was colored. The average age at the time of operation was forty years, with the youngest patient only three months of age and the oldest sixty-two years.

Symptoms: The average duration of symptoms in our 41 cases was three years before admission to the hospital. One patient went for twenty years with a swelling of the temporal region before the underlying meningioma *en plaque* was successfully removed; another went for fifteen years with a unilateral exophthalmos before the tumor was extirpated.

The symptoms produced by the deep, inner or clinoidal group are a unilateral failure of vision associated with a primary optic atrophy which may progress to near-blindness before the other eye becomes involved. On examination of the visual fields, a nasal hemianopsia in the ipsilateral eye is found. A unilateral exophthalmos may develop if the lesion is large enough. If the tumor involves the pituitary fossa, there may be polyuria, polydipsia, and adiposity. The tumors may also affect the carotid artery, the anterior and middle cerebral arteries, and the adjacent dural sinuses.

TABLE IV. CHIEF SYMPTOMS AND SIGNS EXHIBITED BY THE 41 PATIENTS OF OUR SERIES

	CASES
Unilateral exophthalmos	22 (53.6%)
Unilateral visual disturbances not advanced to the stage of blindness	20 (48.8%)
Headache	14 (34.1%)
Unilateral swelling of the temporal region	11 (26.8%)
Diplopia	10 (24.4%)
Bilateral papilledema	10 (24.4%)
Visual field changes	9 (22.0%)
Optic atrophy	9 (22.0%)
Unilateral facial nerve weakness	7 (17.0%)
Unilateral blindness	5 (12.2%)
Diminished corneal reflex on the side of the lesion	5 (12.2%)
Unilateral swelling of eyelids	4 (9.8%)
Unilateral diminished sense of smell	4 (9.8%)
Unilateral papilledema	4 (9.8%)
Ptosis of upper eyelid	2 (4.9%)
Loss of equilibrium	2 (4.9%)

The middle or alar group is often asymptomatic for a long time, and choked disks, more advanced on the side of the lesion, develop before any localizing signs appear. Often the first localizing sign is a contralateral homonymous hemianopsia, usually more advanced in the eye on the side of the lesion. The tumor may grow large enough to cause a unilateral or even a bilateral anosmia.

The outer or pterional group, as stated above, may be divided into *en plaque* and *global* types. The *en plaque* tumors provoke a hyperostosis of the greater and lesser wings of the sphenoid, and the *global* lesions expand within the crotch of the sylvian fissure and slowly push the frontal and temporal lobes apart in a cortical region which is comparatively silent. The typical *en plaque* lesion occurs in women of middle age who for about ten years have observed a slowly increasing unilateral exophthalmos with ultimate impairment of vision and a palpable swelling in the temporal region. Examination reveals edema of the eyelids, proptosis, and a downward displacement of the eye.

The *global* lesions, because they often grow to great size before being recognized, cause symptoms of increased intracranial pressure, such as headache, dimness of vision, and generalized convulsions. Examination shows bilaterally choked disks

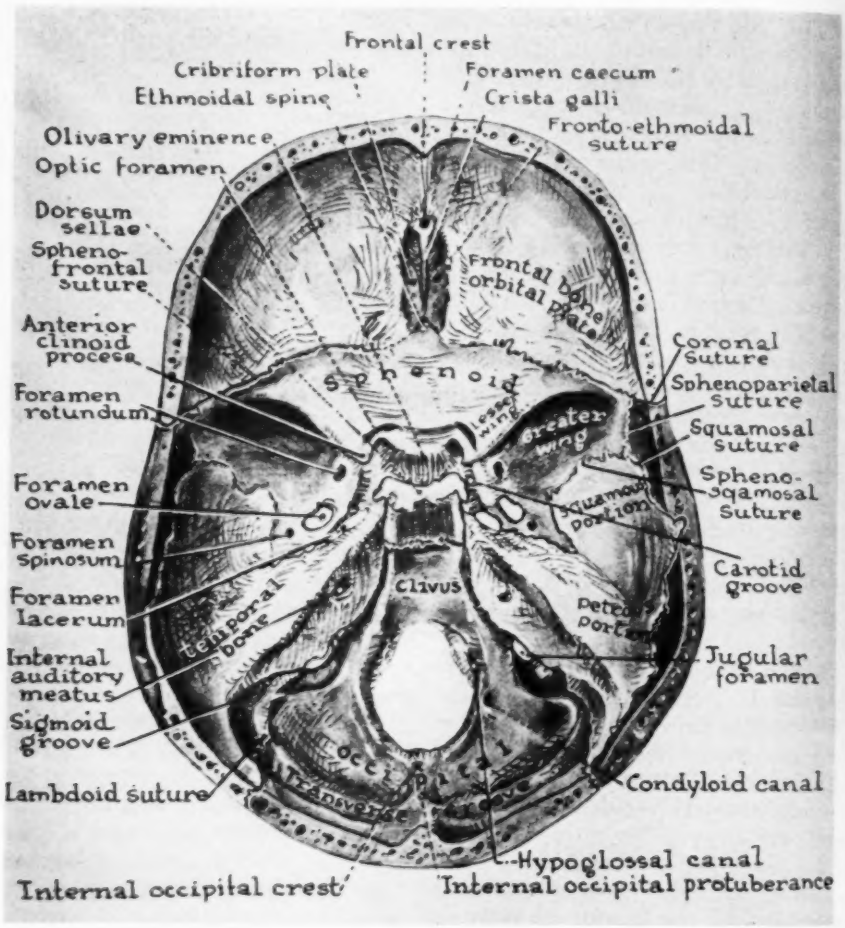

Fig. 3. The ental surface of the base of the skull (*basis cranii interna*). (From Pancoast, Pendergrass, and Schaeffer: The Head and Neck in Roentgen Diagnosis. Courtesy of Charles C Thomas.)

with secondary optic atrophy. Exophthalmos is not common with this type of lesion.

Table IV gives the chief symptoms and signs manifested by the 41 patients in our series. Unilateral exophthalmos and visual disturbances are the most frequent findings, and one should always keep in mind the possibility of a sphenoidal ridge meningioma when examining a patient with these symptoms.

Roentgenologic Diagnosis: In our series of 41 cases, the correct diagnosis was established on 20 patients by a routine roentgen examination of the skull. In another 5 patients, routine skull examination showed evidence of a parasellar mass lesion, which, when operated upon, proved to be a sphenoidal ridge meningioma. With the use of air studies, another 10 patients showed evidence of a parasellar mass lesion. Thus, from plain roentgen studies of the skull and air studies, the correct diagnosis was made on 35 of the 41 patients (85.4 per cent).

Before describing the roentgenologic manifestations of this tumor, an anatomical landmark seen in the base (Hirtz) view of the skull will be discussed. A picture of the internal base of the skull (Fig. 3) is included as an aid in visualizing the shad-

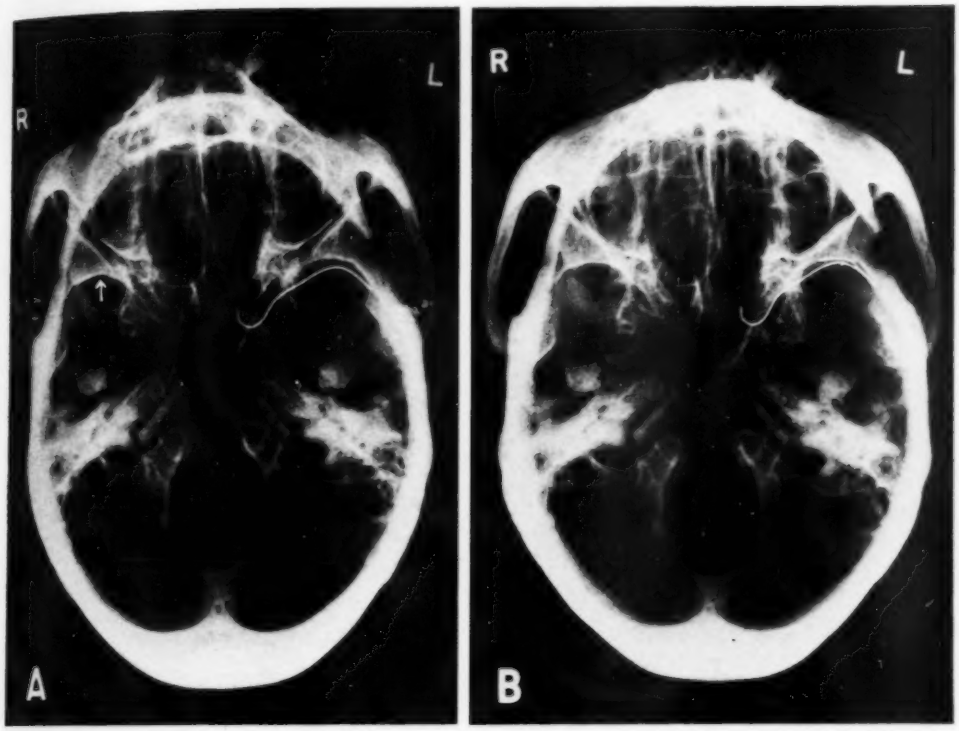

Fig. 4. Base view of a dry skull.

A. The arrow points to the curvilinear shadow, the identity of which we are establishing. The wire is fitted closely around the anterior clinoid and posterior free edge of the lesser wing and extends to pterion.

B. A second wire has been fitted under the lesser wing at the sphenofrontal suture and extends from the lateral border of the superior orbital fissure to pterion. In this illustration a small crosswire is superimposed on the wire hugging the posterior free edge of the lesser wing, and this point is where the lesser wing merges with the greater wing (see Fig. 3).

ows seen in the roentgenogram. The curvilinear shadow seen on both sides, between the anterior and middle fossae (Fig. 4A), has at times been described as representing the lesser wing of the sphenoid. In order to establish the identity of this shadow, a wire was fitted as closely as possible to the posterior free edge of the anterior clinoid and lesser wing of the sphenoid and carried beyond the junction of the lesser and greater wings to the pterion. It is obvious (Fig. 4A) that the curvilinear shadow in question is in no way superimposed on the lesser wing of the sphenoid. A second wire was then fitted under the lesser wing, at the sphenofrontal suture, extending from the lateral border of the superior orbital fissure to the pterion. It can be seen (Fig. 4B) that the curvilinear shadow is superimposed on the second wire. There were also cut numerous sections from the sphenoidal ridge in order to visualize an interruption of the curvilinear shadow. These are not illustrated, but it has been concluded that the shadow seen on the roentgenogram represents the anterior-superior surface of the greater wing of the sphenoid in the region of the sphenofrontal suture.

The roentgen signs of a meningioma of the inner third are hyperostosis or increased density of the ipsilateral anterior clinoid, the optic canal, and inner portion of the lesser wing of the sphenoid, seen best in the postero-anterior and optic canal view (Fig. 5). Occasionally, the increased density of the hyperostosis can be seen in the Hirtz and lateral stereoscopic projections.

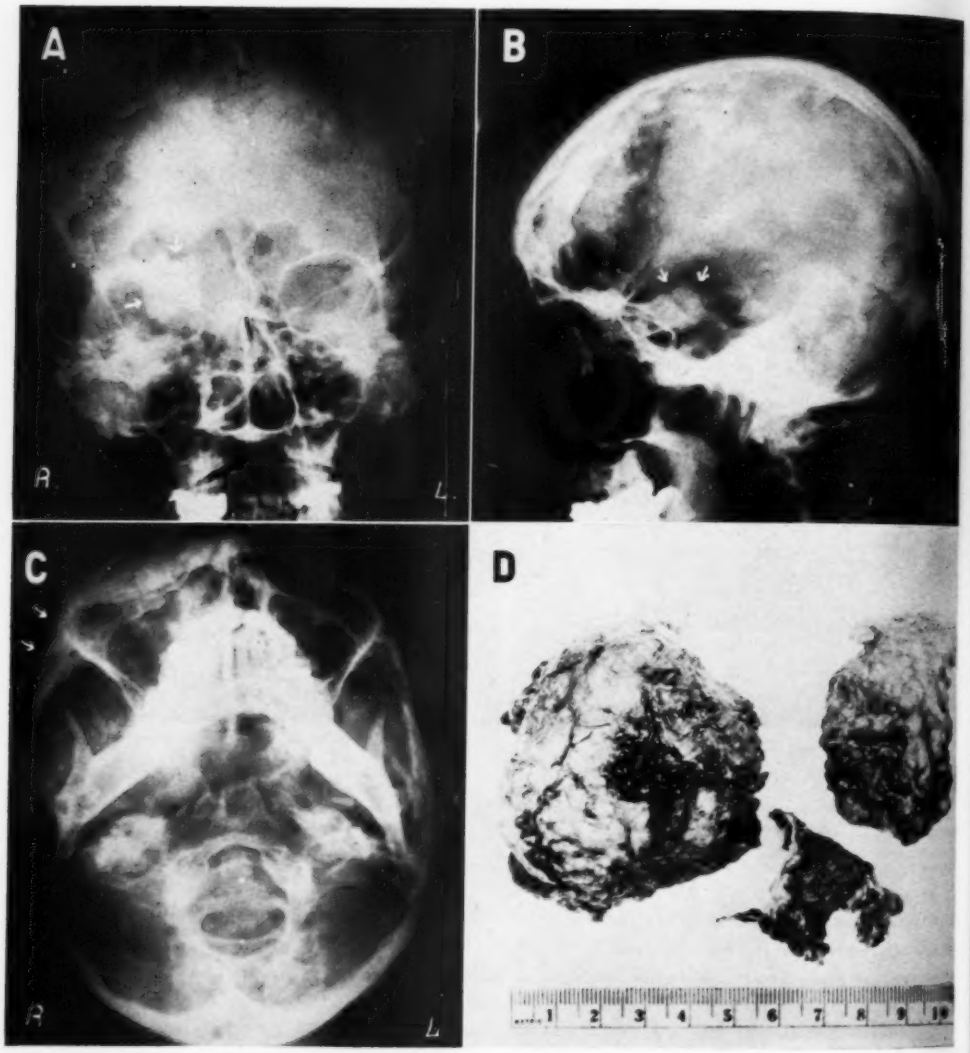

Fig. 5. Case 16: A 32-year-old white female developed a rhinorrhea and had a growth removed from her right nostril on two different occasions. Subsequently frontal sinusitis developed, and on opening the frontal sinus a growth was seen coming through the posterior wall of the sinus. Eight months after the onset of her symptoms she was referred to the Hospital of the University of Pennsylvania and for the first time roentgenograms were taken. A diagnosis of a meningioma of the right sphenoidal ridge was made.

A. Postero-anterior view showing a 2 × 2 cm. area of calcification in the region of the lesser wing of the sphenoid on the right side (arrows); the right ethmoid and frontal sinus are clouded.

B. Right lateral view showing the calcification lying over the planum sphenoidale and sella turcica (arrows). The anterior clinoid processes are blunted and the dorsum sellae is eroded. There is also considerable scalloped thickening of the inner table of the right frontal bone.

C. Base view showing thickening of the right frontal, maxillary and ethmoid sinuses and an area of increased density at pterion (arrows). Note how the curvilinear shadow described in Fig. 4 is missing on the right side.

D. Photograph of the resected tumor and part of the sphenoidal ridge. (Courtesy of Armed Forces Institute of Pathology, Accession Number: 218242-6.)

Quite frequently it has been found that lateral stereoscopic roentgenograms of the contralateral side will show the hyperostosis better than similar roentgenograms of the ipsilateral side (Figs. 6, 7, and 8). If bone erosion is present, one must be careful

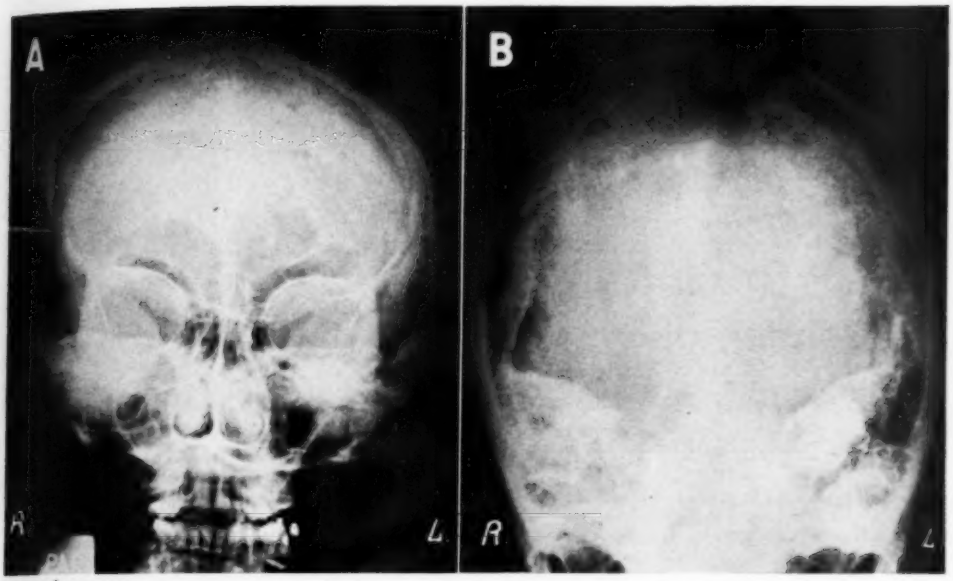

Fig. 6. Case 32: A 42-year-old white female had a draining right ear for thirty-seven years and a failure of vision and diplopia for three months. Examination showed bilateral papilledema with 3 diopters choking on the right and 1 diopter on the left, left central facial weakness, increased deep reflexes on left, questionable left hemiparesis, and definite torpor and mental deficiency. Roentgenograms were interpreted as negative except for a sclerotic right mastoid. A plunge for a suspected abscess on the right side was done, but no abscess was found. The patient suddenly expired the night before a contemplated craniotomy.
A. Postero-anterior view. In retrospect, some increased density of the right greater and lesser wings of the sphenoid is discernible. This was missed originally and shows the importance of evaluating slight differences in density seen through the orbits. B. Occipital view showing a sclerotic right mastoid. C (below). Base view showing nothing abnormal.

to exclude a normal variant, an aneurysm, or a glioma (Fig. 9). If the lesion cannot be demonstrated by changes in the bone or conventional roentgenograms, it can be demonstrated readily by encephalography, ventriculo-encephalography, or ventriculography (Fig. 10).

The middle ridge tumors may show hyperostosis or bone absorption of the lesser and greater wings of the sphenoid. A non-Bucky frontal sinus view to show changes in the sphenoidal ridges seen through the orbits (Figs. 11, 12, and 13) is extremely valuable. If bone absorption of the ridge is present, one should be careful to exclude the normal variant of a ridge with either decreased density or one that has a dehiscence-like appearance.

The pterional *en plaque* sphenoidal ridge tumors produce very pronounced hyperostosis of the greater and lesser wings of the sphenoid, and in some instances of the

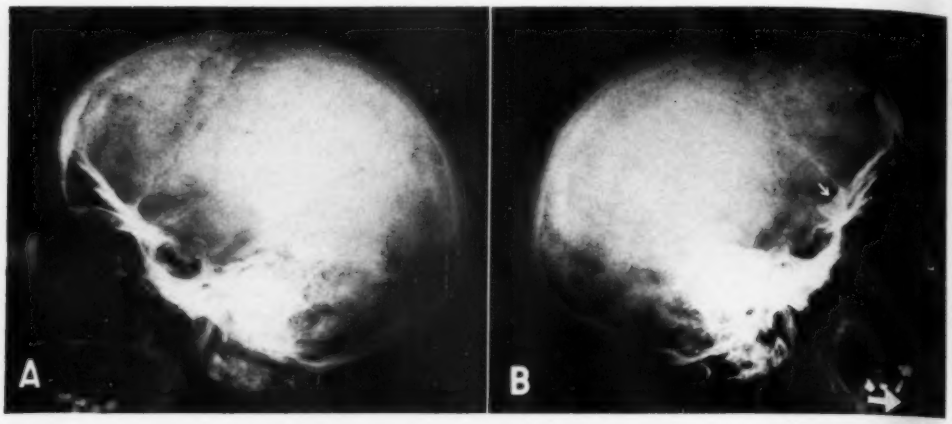

Fig. 7. Case 32, shown also in Fig. 6.
A. Right lateral view showing nothing abnormal.
B. Left lateral view showing a very definite increased density above the planum sphenoidale which in stereo projects along the right sphenoidal ridge (arrow). This was missed, or at least not evaluated, because all the attention was drawn to the right lateral view. This demonstrates the lesion showing up on the roentgenogram of the contralateral side.

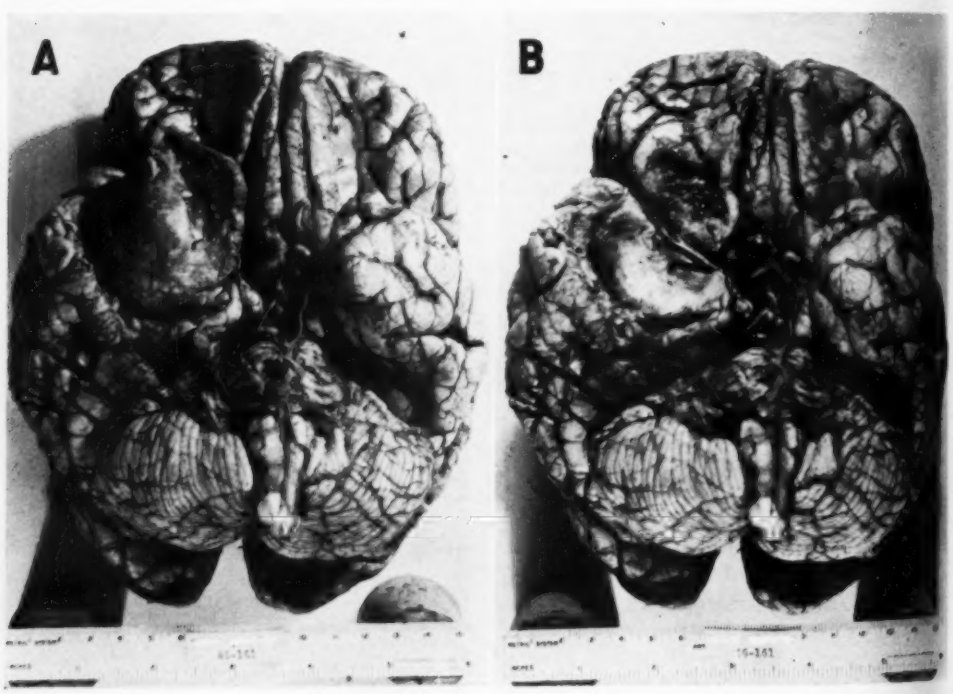

Fig. 8. Case 32, shown also in Figs. 6 and 7.
A. Autopsy specimen showing the tumor embedded in the right temporal lobe.
B. Autopsy specimen showing the tumor bed between the frontal and temporal lobes.

orbital plate of the frontal, the squamosal portion of the temporal, and the pterional region. In the postero-anterior roentgenograms, with the shadows of the greater and

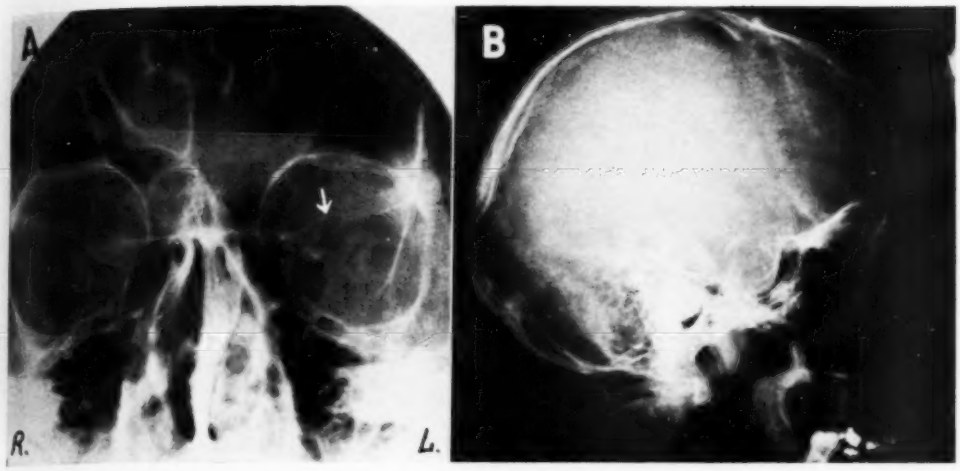

Fig. 9. Case 15: A 25-year-old white male with a five-year history of left frontal sinusitis and edema of left eyelids. Two months prior to admission he noticed a left-sided exophthalmos, blurring of vision of his left eye, and left-sided supraorbital pain. Operation revealed a sphenoidal ridge meningioma on the left side.
A. Frontal sinus view showing slight erosion of the lesser wing of the sphenoid on the left side (arrow).
B. Left lateral view showing an enlarged hypophyseal fossa which measured 16 mm. in anteroposterior diameter and 16 mm. in depth.

Fig. 10. Case 23: A 62-year-old white male had loss of equilibrium, nausea, loss of memory, and headache of five weeks duration. Examination showed bilateral papilledema which was more pronounced on the right, positive Romberg with fall to the right, slight left facial paralysis, and electro-encephalographic changes indicative of a lesion in the right frontal lobe. A routine roentgenogram of the skull showed nothing abnormal. A postero-anterior ventriculogram showed failure of the right lateral ventricle to fill and displacement of the left lateral and third ventricles to the left. In the lateral view (not shown) the posterior portion of the third ventricle was not filled. At operation a sphenoidal ridge meningioma on the right side was removed.

lesser wings of the sphenoid projected into the orbit, their eburnated appearance as compared with the opposite side is pathognomonic (Fig. 14). One can also recognize the unique eburnated changes in the lateral stereoscopic views, particularly in the region of pterion (Fig. 15). This type of lesion is to be differentiated from leontiasis ossea and osteitis deformans (Fig. 16). Benign osteomas seldom cross suture lines, whereas it is rare to have a meningioma confined to a single bone in the pterional region.

The pterional *global* tumors produce increased vascularity—multiple vascular grooves—in the pterional region (Figs. 17 and 18). These are best seen in lateral stereoscopic roentgenograms. In the larger global tumors, the pituitary fossa may be so deformed, that one may suspect that the lesion has either arisen within or about the fossa (Fig. 19).

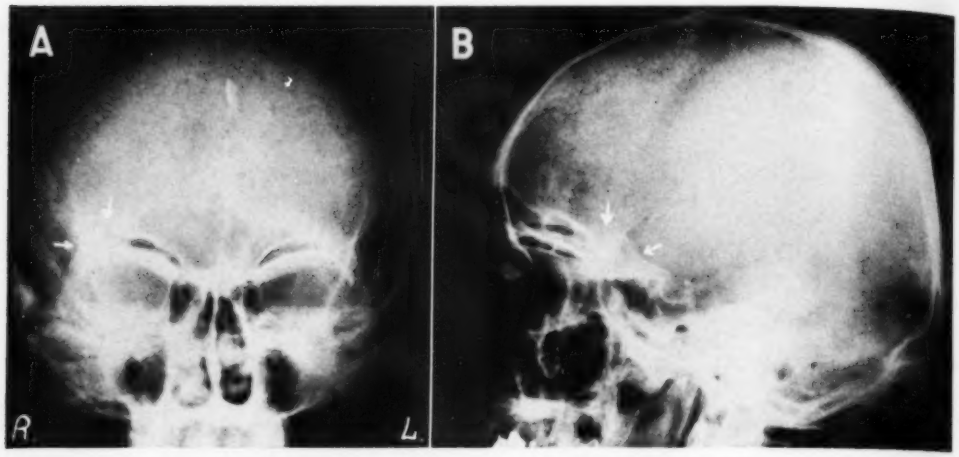

Fig. 11. Case 11. A 46-year-old white male, twenty months before admission to the hospital, experienced a severe pain in the right frontal region. Five months later he began to have generalized convulsions and shortly before admission he had an attack in which his left hand began to shake prior to loss of consciousness. Examination showed a slight tremor of hands, slight bilateral exophthalmos, and impaired olfactory sense on the right. At operation a large sphenoidal ridge meningioma was removed from the right side.

A. Postero-anterior view showing an increased density of the entire right sphenoidal ridge, most pronounced in the outer third (arrows).

B. Right lateral view showing an increased density of the right sphenoidal ridge (arrows).

One of our cases (Case 28) produced considerable erosion of the lateral wall of the orbit, the adjacent frontal bone, the greater wing of the sphenoid bone and, to a lesser extent, the lesser wing of the sphenoid bone. Perpendicular spicules of bone radiated into the soft-tissue mass projecting out from the frontal bone. The whole picture was that of an osteogenic sarcoma, and yet the pathological diagnosis was a meningioma Type III, variant 4 (Fig. 20).

Postoperative Survival: Two patients died preoperatively and 1 of immediate postoperative complications. One patient died within two weeks from bronchopneumonia, and 1 within two months from pulmonary complications. Three died within about six months as a result of continued growth of the tumor, 2 between three and six years, and 1 after fourteen years. One died of a cerebrovascular accident at approximately twelve years. One died at about eight months, cause unknown. On 2 patients there was inadequate follow-up. The remaining 26 patients are alive for eighteen years to less than a year for those operated on in 1948. Certainly the prognosis of this tumor is good, and as the diagnosis can be made in 85 per cent of the cases by the radiologist, it behooves us all to know the roentgen findings of this most interesting tumor.

SUMMARY

A review of 41 verified sphenoidal ridge meningiomas shows that the diagnosis was correctly established by roentgen findings in 35 of the cases (85.4 per cent). Following complete surgical removal, a good prognosis can be offered these patients. The patients in this series averaged three years after the onset of symptoms before reaching the hospital. This long delay should be avoided, if possible, because the earlier lesions are more easily resected.

This discussion has been presented to acquaint radiologists and others who might be interested in the characteristic symptomatology and roentgen findings of this tumor. A unilateral exophthalmos, visual disturbances, and a swelling in the temporal region should always bring to mind the possibility of a sphenoidal ridge meningioma. When this characteristic symptom-

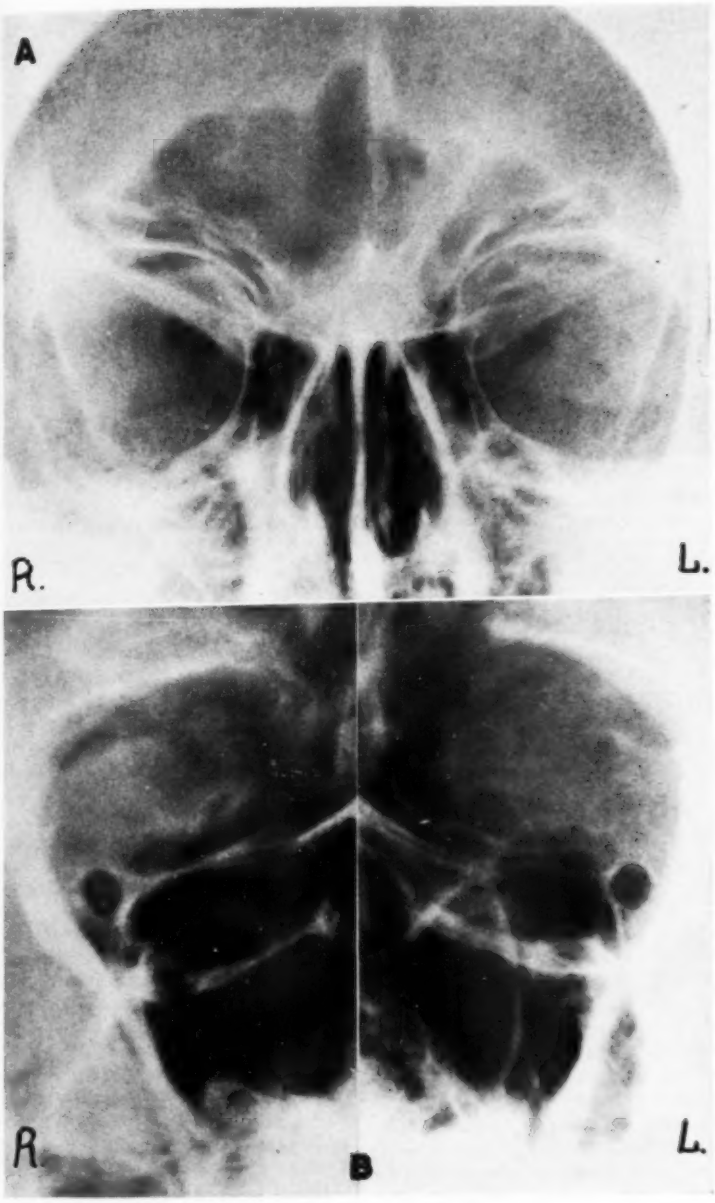

Fig. 12. Case 11, shown also in Fig. 11.

A. Non-Bucky frontal sinus view showing the density of the outer third of the right sphenoidal ridge.

B. View of the optic foramina showing an increased density of the wall on the right side.

atology is associated with hyperostosis of the lesser or greater wings of the sphenoid, a positive diagnosis can usually be made.

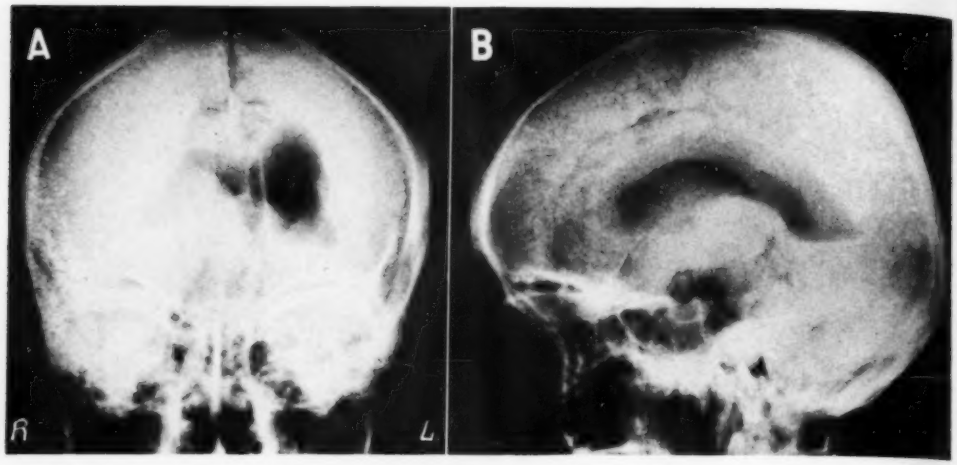

Fig. 13. Case 11, shown also in Figs. 11 and 12.
A. Postero-anterior encephalogram showing displacement of both lateral and third ventricles to the left; the left lateral ventricle is slightly enlarged. The anterior half of the right lateral ventricle is encroached on by a mass on the right side.
B. Lateral encephalogram showing encroachment from below on the anterior horn of the right ventricle.

CASE HISTORIES

CASE 1: M. A., age 36, white female, admitted 6-30-30.

Chronology of Symptoms: Impaired vision of right eye in 1927. In 1929, vision of left eye also became impaired and headache began in vertex and parietal regions. Vertigo for five months prior to admission.

Physical and Neurologic Findings: Bitemporal hemianopsia; waxy pallor of both disks. Poor vision, worse on right side. Spinal fluid pressure: 300 mm. of water.

Roentgen Findings: Slight thinning of the dorsum sellae. Sella turcica top-normal in size. Anterior-posterior measurement 12 mm., depth 8 mm. Examination suggestive of pituitary tumor.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Suprasellar lesion such as a craniopharyngioma or suprasellar meningioma.

Operative Procedure (7-7-30): Transfrontal craniotomy on right side, with exposure and partial removal of right-sided sphenoidal ridge meningioma. On 8-2-30, a second operation with removal of more of the tumor.

Postoperative Course: Stormy, but patient finally discharged on 9-4-30.

Pathologic Findings: Tumor removed piecemeal. Meningioma Type II, variant 1.

Survival Period: Eighteen years. Living and well, 3-12-48, but completely blind.

CASE 2: W. B., age 36, white male, admitted 2-16-31.

Chronology of Symptoms: Double vision first noticed in August 1930, and right pupil became larger than left. Onset of right frontal headaches in December 1930, more severe in recumbent position. Three weeks prior to admission, the right eyelid began to droop; two weeks before admission it was completely drooped.

Physical and Neurologic Findings: Ptosis of right eyelid. Right pupil is larger than left and does not react to light and accommodation. Right eye can only be moved upward. Left homonymous hemianopsia.

Roentgen Findings: Pituitary fossa top-normal in size. Anterior-posterior measurement 12 mm.; depth 11 mm. Dorsum sellae intact but rarefied.

Ventriculographic Findings (3-3-31): Right lateral ventricle is not as large as left and its lumen is encroached upon from the side and below. Appearance suggests a tumor in the middle fossa on the right side.

Encephalographic Findings (2-23-31): Right lateral ventricle failed to fill, but the left lateral ventricle, third ventricle, and fourth ventricle were visualized. Third ventricle deviated to left.

Preoperative Diagnosis: Tumor in the right middle fossa.

Operative Procedure (3-6-31): Transfrontal flap elevated, with exposure and partial removal of a tumor lying on the inner third of the right sphenoidal ridge just outside the internal carotid artery.

Postoperative Course: Patient made good recovery, but died suddenly of cerebrovascular accident 12-6-43. The paralysis of the right third nerve had almost completely disappeared.

Pathologic Findings: Meningioma Type I, variant 3.

Survival Period: 12 years, 9 months.

CASE 3: M. S., age 32, white female, admitted 10-16-31.

Chronology of Symptoms: Difficulty with vision beginning four years prior to admission. Complete loss of vision in right eye, and gradual loss of considerable vision of left eye over the four years. Headaches developed, which caused patient to seek medical aid.

Physical and Neurologic Findings: Blindness of right eye; optic atrophy of right eye. Papillitis of left eye. Temporal hemianopsia on the left.

Roentgen Findings: Posterior clinoids and dorsum sellae completely eroded. Floor of sella thin, encroaching on sphenoidal sinus.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Parasagittal tumor on the right, probably a meningioma.

Operative Procedure (11-16-31): Right transfrontal craniotomy with exposure and partial removal of a meningioma of right sphenoidal ridge.

Postoperative Course: Patient died eight hours postoperatively, due to hemorrhage.

Pathologic Findings: Meningioma Type II, variant 3.

CASE 4: E. G., age 40, white female, admitted 2-6-33.

Chronology of Symptoms: Double vision first developed in November 1932. Since then, patient has had some blurring of vision and sees better if she reads with only her right eye. Visual hallucinations.

Physical and Neurologic Findings: Five diopters choking in left eye. Decreased vision in left eye. Weakness of right side of face. Positive Hoffman's sign, right hand. Deviation of tongue to right. Spinal fluid pressure:350 mm. of water. Deviation of uvula to right. Decreased sense of smell on right.

Roentgen Findings: Erosion of floor of sella on left side. Erosion of dorsum sellae. Occipital view shows dorsum eroded on left side. Anterior-posterior measurement 8 mm. on right side and 11 mm. on left side; depth 8 mm. on right and 11 mm. on left side. These changes indicate a left cerebral tumor, probably frontal or frontotemporal. Optic foramina views show complete absence of the upper half of right foramen.

Ventriculographic Findings: Ventricles shifted to the left. Left lateral ventricle normal in size and shape. Right lateral ventricle deformed by pressure exerted from the right side. Absence of the anterior horn on the right side.

Encephalography: Not done.

Preoperative Diagnosis: Prior to ventriculography, it was thought that a mass lay in the left frontotemporal region, but the ventriculogram placed the lesion on the right side.

Operative Procedure (2-15-33): Right transfrontotemporal craniotomy, with exposure and complete removal of a large sphenoidal ridge meningioma.

Postoperative Course: Complete recovery except for a blind spot in right visual field.

Pathologic Findings: Specimen of tumor weighed 144 gm., and the largest nodule measured 5 × 6 × 4 cm. Meningioma Type III, variant 3.

Survival Period: 15 years, 4 months. Living and well, July 1948.

CASE 5: V. B., aged 45, white female, admitted 7-6-33. (Fig. 15)

Chronology of Symptoms: Twenty-year history of a swelling of the right temporal region. Exophthalmos of right eye for five years. Just prior to admission some mental changes observed by family.

Physical and Neurologic Findings: Hard bony swelling in right temporofrontal region. Exophthalmos of right eye; optic atrophy, secondary, of right eye, with poor vision.

Roentgen Findings: Large dense bony mass involving the right side of the skull in temporofrontal region. Lesion involves the roof and lateral wall of the orbit, and both the lesser and greater wings of the sphenoid. Lateral view shows the area of bony involvement to be about 10 cm. in diameter, centering in the pterion.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (8-4-33): Right transfrontal craniotomy with removal of a large osteoma with an underlying tumor which involved the floor of the anterior fossa and the sphenoidal ridge and extended back into the middle fossa.

Postoperative Course: Favorable.

Pathologic Findings: Bony hyperostosis measured 7.5 cm. in thickness and 8.5 cm. in diameter. Underlying the hyperostosis was a meningioma *en plaque*, Type III, variant 3.

Survival Period: 1 year, 7 months. Living when last heard from, March 1935.

CASE 6: H. S. C., age 41, white male, admitted 10-7-33.

Chronology of Symptoms: Severe frontal and occipital headaches with diplopia developed in winter of 1932. Ethmoid and sphenoid sinuses were drained in January 1933 and the cephalgia disappeared. Head colds in September 1933, followed by return of headache in left frontotemporal region. On 10-3-33 diplopia again developed. On 10-4-33 patient noted a marked diminution in vision and began to vomit and become lethargic.

Physical and Neurologic Findings: Patient lethargic. Slow pulse (50 to 60). Sluggish pupillary reaction to light, accommodation, and convergence; left external rectus palsy. Slight dysmetria of left hand. Suggestion of right lower facial weakness. Loss of sense of smell. Impaired vision

with bilateral choking of disks of 2 diopters. Positive Babinski on left. No visual field obtained with left eye and markedly contracted visual field with right eye.

Roentgen Findings: Pituitary fossa enlarged. Anterior-posterior diameter 13 mm.; depth 12 to 15 mm. Anterior clinoid processes thinned; slight atrophy of the dorsum sellae. Floor of fossa thinned and deeper on one side than the other. Both ethmoid regions clouded; thickened mucous membrane in both maxillaries.

Ventriculographic Findings: Postero-anterior view showed evidence of a mass lesion on the left side, encroaching on left lateral ventricle and pushing both lateral ventricles to right. Third ventricle also shifted to the right. Both lateral ventricles slightly dilated. Third ventricle encroached upon from below and posteriorly.

Encephalography: Not done.

Preoperative Diagnosis: Prior to ventriculography it was thought the patient had a left frontal abscess. After ventriculography the diagnosis was a temporal lobe mass lesion on left side.

Operative Procedure: On 10-11-33 needle exploration for a left frontal abscess was negative. On 10-14-33 a left temporoparietal craniotomy was performed, with exposure and removal from the left temporal region of a meningioma originating from the outer portion of the sphenoidal ridge.

Postoperative Course: Good recovery, but no return of vision.

Pathologic Findings: 70 gm. of tumor tissue removed, size of an egg. Meningioma Type II, variant 1.

Survival Period: 11 years known survival. Living and well, except for blindness, when last heard from in 1944.

CASE 7: R. A., age 26, colored female, admitted 11-14-33.

Chronology of Symptoms: One year prior to admission patient noticed her right eye was prominent, and two months prior to admission that it was bulging. No other symptoms except occasional scotomata after using the eyes for a long time.

Objective Findings: Unilateral exophthalmos of right eye.

Roentgen Findings: Increased density of right sphenoidal ridge; enlargement of sella turcica. Encroachment on right optic foramen.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Right-sided sphenoidal ridge meningioma.

Operative Procedure (12-7-33): Right transfrontal craniotomy with exposure and verification of a tumor involving the sphenoidal ridge on the right side. The tumor extended from the outer third all the way into the inner third. Not much of it could be removed.

Postoperative Course: Patient was started on roentgen therapy, 10-13-34, because of progression of symptoms. The right eye was removed on 5-20-43 and a large mass of meningioma tissue was removed from the back of the orbit. Patient died 2-10-47.

Pathologic Findings: Meningioma Type III, variant 1.

Survival Period: 13 years, 2 months.

CASE 8: O. McG., age 36, white female, admitted 1-13-34.

Chronology of Symptoms: Loss of vision in left eye noticed in December 1932, beginning in upper and outer quadrants of visual field. By July 1933 patient was almost blind in left eye. In December 1933, began to notice loss of vision in right eye. Frontal headaches for one year. Visual hallucinations, left eye, for several months.

Physical and Neurologic Findings: Primary optic atrophy, left optic disk; blindness in left eye except for superior nasal quadrant. Marked temporal cut in right eye. Hyperactive reflexes in upper extremities.

Roentgen Findings: Some increased intracranial pressure shown by appearance of the pituitary fossa. Anterior-posterior measurement 12 mm., depth 9 mm. Posterior-anterior view shows some calcification in middle fossa on left. Roentgenograms remarkable in showing so few changes.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Tumor in left frontal and frontotemporal region.

Operative Procedure (1-19-34): Left transfrontal craniotomy, with partial removal of a sphenoidal ridge meningioma. Operation stopped because of bleeding. 1-22-34, a second stage carried out, with decompression by removal of bone flap. Planned to remove rest of tumor at a later date.

Postoperative Course: Stormy. Motor aphasia of right arm developed. Patient died 7-12-34.

Pathologic Findings: Only a few pieces of tissue removed. Meningioma Type II, variant 2.

Survival Period: 6 months.

CASE 9: H. E., age 42, white male, admitted 11-10-34.

Chronology of Symptoms: Dull aching pain developed in left arm in April 1931, lasting for five months. In December 1931, steady aching pain over left malar region and nasal ala developed; disappeared in April 1932. In October 1932 patient began to be easily fatigued and lost 20 lb. In July 1933, blurred vision, diplopia, and pain in region of left eyeball developed. Gradually left eye became proptosed. December 1933, pain in left eye severe; roentgenogram revealed an osteoma of the lateral, superior, and inferior walls of the orbit. A decompression of the orbital roof was performed and nothing more than thickened bone was found. Vision gradually failed and in April 1934 further medical aid was sought, but no diagnosis was made.

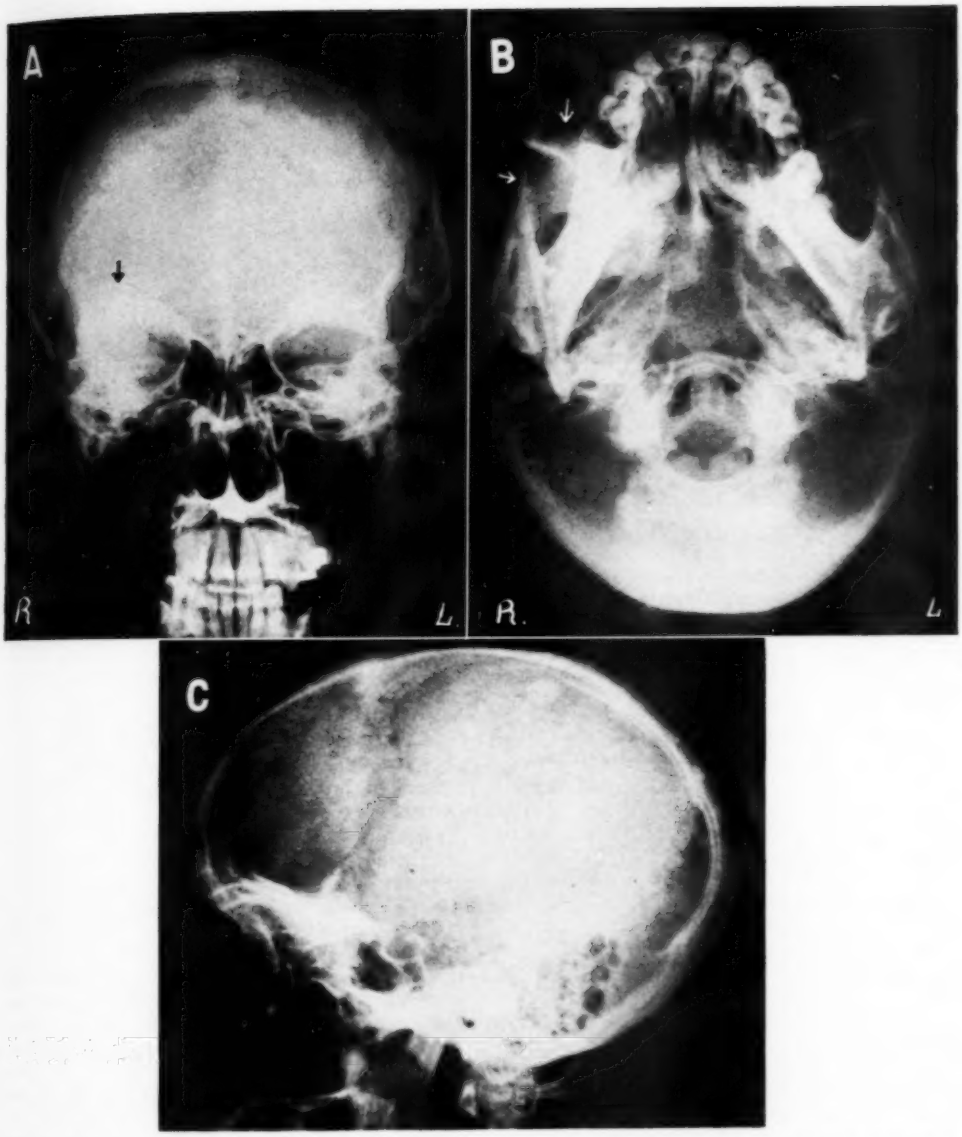

Fig. 14. Case 14: A 36-year-old white female had for eight months noticed a "pressure" in the occipital region and prominence of the right eye. Examination revealed an exophthalmos of the right eye, diminution of pain recognition in the right side of the face, a deviation of the tongue to the right, and a weakness of the right masseter. Operation revealed a sphenoidal ridge meningioma on the right side.

A. Postero-anterior view showing hyperostosis of both the greater and lesser wings of the sphenoid on the right side.

B. Base view showing the hyperostosis to occupy the region of the curvilinear shadow shown in Fig. 4A. The shadow has been completely lost in the hyperostosis, which has spread medially and posteriorly into the temporal fossa.

C. Lateral view showing the hyperostosis to involve the greater and lesser wings of the right side of the sphenoid and also the superior orbital plate on the right side.

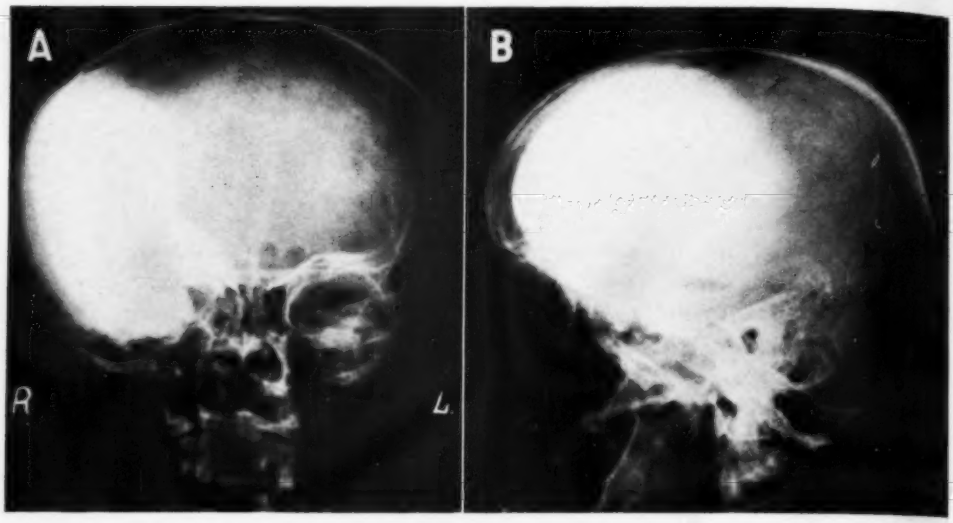

Fig. 15. Case 5: A 45-year-old white female had a slowly growing hard bony swelling in the right temporofrontal region for twenty years. In recent years, prior to her admission, she had noticed exophthalmos of the right eye and experienced severe headaches and blurred vision. Examination showed the swelling in the right frontotemporal region and exophthalmos of the right eye, with poor vision. At operation a sphenoidal ridge meningioma *en plaque* was removed along with the hyperostosis.

A. Postero-anterior view showing a large dense bony mass involving the right temporal, frontal, and sphenoid bones.

B. Right lateral view showing the large dense bony mass involving the right frontal, temporal, parietal, and sphenoid bone.

Physical and Neurologic Findings: Pain distributed to area supplied by left maxillary division of the 5th nerve. Left palpebral fissure narrowed; impairment of rotation of left eye; exophthalmos of left eye; optic atrophy on the left; impaired corneal reflex on the left. Right pupil reacted to direct light but not to indirect light. Left pupil reacted to indirect but not to direct light.

Roentgen Findings: Destruction of the left sphenoidal ridge.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Left sphenoidal ridge meningioma.

Operative Procedure (11-20-34): Frontotemporal exploration, with exposure and removal of a tumor from the lesser wing of the left sphenoid, which completely surrounded the left optic nerve.

Postoperative Course: Wound healed but patient continued to have much pain in region of left eye. In 1944 he was operated on again at Johns Hopkins Hospital and a large intraorbital growth was removed, as well as the left eye.

Pathologic Findings: Meningioma Type III, variant 2.

Survival Period: 10 years known survival. No record since 1944.

CASE 10: B. F., age 34, white female, admitted 2-18-35.

Chronology of Symptoms: Edema of left upper eyelid of eight months duration. Swelling of left frontotemporal region for six months. Protrusion of left eye for three months. No subjective visual disturbances.

Physical and Neurologic Findings: Hard, fixed, irregular, painless mass, 3 cm. in diameter, above and behind the temporal end of the left supraorbital ridge. Left-sided exophthalmos. Sense of smell diminished on left.

Roentgen Findings: Wing of the sphenoid on left side destroyed and replaced by a large irregular area of increased density extending medially to the nasal fossa and laterally as far as the outer canthus of the eye.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on left side.

Operative Procedure (3-8-35): Left transfrontal craniotomy with exposure and removal of an osteoma and a small meningioma underlying it within the dura.

Postoperative Course: Favorable. Patient last heard from in July 1944, at which time she had no symptoms.

Pathologic Findings: Tumor and dura weigh 4 gm.; measure 2.5 × 1.5 cm. The hyperostosis weighs 62 gm. Meningioma Type II, variant 2.

Survival Period: 10 years known survival.

CASE 11: R. M., age 46, white male, admitted 8-14-35. (Figs. 11–13)

Chronology of Symptoms: Severe pain in right frontal region in January 1934. In May 1934, "spell" of unconsciousness with frothing at mouth, glassy stare, and rigid limbs. In June 1935 patient experienced his fifth attack and noticed that his left hand began to shake prior to loss of consciousness.

Physical and Neurologic Findings: Slight tremor of fingers of left hand. Slight bilateral exophthalmos. Hypoactive abdominal reflexes. Impaired olfactory sense on right.

Roentgen Findings: Increased density of entire right sphenoidal ridge, most pronounced in outer third. Views of the optic foramina show increased density of the wall on the right side.

Ventriculography: Not done.

Encephalographic Findings: Lateral ventricles displaced slightly to left. Left lateral ventricle slightly enlarged and right lateral ventricle distorted by a mass lesion on right side.

Preoperative Diagnosis: Mass lesion in region of right sphenoidal ridge and temporal lobe.

Operative Procedure (8-30-35): Right frontotemporal craniotomy, with exposure and removal of a large sphenoidal ridge meningioma. One-third of the tumor lay in the anterior fossa and the remainder in the middle fossa.

Postoperative Course: Bronchopneumonia developed and patient died 9-13-35.

Pathologic Findings: Tumor weighed 54 gm. Meningioma Type I, variant 2.

Survival Period: 2 weeks.

CASE 12: E. M., age 43, white female, admitted 5-27-36.

Chronology of Symptoms: Failing vision noticed in summer of 1934. In December 1934, blindness in right eye, vision impaired in left. Severe periodic occipitofrontal headaches began early in 1935.

Physical and Neurologic Findings: Bilateral optic atrophy; right pupil does not react to direct light and left pupil does not react to consensual light. Impaired vision in left eye, with marked concentric field contraction. Loss of sense of smell on right.

Roentgen Findings: No evidence of increased intracranial pressure. Pituitary fossa enlarged, with evidence of erosion of dorsum sellae and posterior clinoids, and some thinning of the floor of the fossa. Anteroposterior diameter 17 mm.; depth 12 mm. Pineal in midline.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Suprasellar or intrasellar lesion.

Operative Procedure (6-3-36): Right transfrontal craniotomy, with removal of tip of right frontal lobe and biopsy of a right sphenoidal ridge meningioma. On 6-8-36, a second stage was performed, with partial removal of the tumor.

Fig. 16. Case of fibro-osseous dysplasia in a 57-year-old white female which was first thought to be a meningioma.

Postoperative Course: Patient recovered from the operation but remained blind in the right eye. Gradually went down hill and died 10-30-42.

Pathologic Findings: Meningioma Type 2, variant 2.

Survival Period: 6 years, 4 months.

CASE 13: C. H., age 38, white female, admitted 1-23-37.

Chronology of Symptoms: Dull aching pain in eyeballs for two years. Pain in the face and head of one years duration. Difficulty in vision, especially in right eye, for six weeks.

Physical and Neurologic Findings: Bilateral choked disks with hemorrhages and exudates. Right biceps and triceps and abdominal reflexes more active than the left. Choked disk of 7 diopters on right and 7 diopters on left.

Roentgen Findings: Negative.

Ventriculographic Findings: Lateral and third ventricles displaced to left.

Encephalography: Not done.

Preoperative Diagnosis: Right cerebral tumor.

Operative Procedure (1-29-37): Right temporofrontal occipital craniotomy, with exposure and removal of meningioma of the sphenoidal ridge.

Pathologic Findings: Meningioma measuring 5× 5.5 × 3 cm., Type II, variant 1.

Survival Period: 12 years, 1 month. Well 2-2-49.

CASE 14: B. L. C., age 36, white female, admitted 6-14-37. (Fig. 14)

Chronology of Symptoms: For many years suffered from temporal headaches. For past eight months noticed "pressure" in the occipital region and a prominence of the right eye.

Physical and Neurologic Findings: Exophthalmos of right eye, with slight asymmetry of right side of face. Diminution of pain recognition, right side of face. Tongue deviated to right. Weakness of right masseter muscle.

Roentgen Findings: Bony hyperostosis of greater wing of sphenoid and orbital plate of frontal bone on the right side.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Right-sided sphenoidal ridge meningioma.

Operative Procedure (7-10-37): Right transfrontal craniotomy, with exposure and removal of a meningioma involving the roof and outer wall of the orbit and the right sphenoidal ridge, together with a meningioma *en plaque* involving the dura at the outer end of the sphenoidal ridge.

Postoperative Course: January 1937, roentgen studies showed an increased density and thickening of the lateral wall of the orbit with narrowing of the right orbital canal. Proptosis of the right eye began in June 1946. On 3-19-47 roof of right orbit was removed.

Pathologic Findings: Meningioma *en plaque*, 3 × 4 × 0.5 cm. thick, Type III, variant 2.

Survival Period: 12 years, 3 months. Living and well.

CASE 15: I. S., age 25, white male, admitted 9-14-37. (Fig. 9)

Chronology of Symptoms: Diabetes for eight years. Five-year history of left frontal sinusitis and swelling of eyelids. Loss of 15 lb. in five months. Exophthalmos for two months. Impaired vision in left eye for ten days. Supraorbital pain for two days.

Physical and Neurologic Findings: Exophthalmos of left eye, with globe turned down and to the left. Swelling of left eyelid. Impaired convergence of left eye. Diplopia on looking upward and to the left (superior rectus). Diminished corneal reflex.

Roentgen Findings: Hypophyseal fossa enlarged: anteroposterior measurement 16 mm.; depth 16 mm. Slight erosion of left sphenoidal ridge. Multiple sinus disease with fluid in right antrum. Some erosion of left optic foramen.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on left side.

Operative Procedure (9-23-37): Left transfrontal craniotomy and partial removal of a tumor lying in the sella turcica, extending from the left sphenoidal ridge.

Postoperative Course: Osteomyelitis of bone flap developed; flap removed 1-22-38. Patient felt well following bone flap removal. Died in December 1945. No autopsy; cause of death unknown.

Pathologic Findings: Specimen consisted of a few small pieces of soft tissue, diagnosed as meningioma Type I, variant 4.

Survival Period: 8 years, 3 months.

CASE 16: B. B., age 32, white female, admitted 10-31-37. (Fig. 5)

Chronology of Symptoms: Rhinorrhea of right nostril developed in February 1937, and a physician removed a growth from the nose. It grew, and was again removed a week later. A frontal sinusitis and an acute otitis media developed. Right eye began to bulge and another physician found 4½ diopters choking bilaterally; he opened the right frontal sinus to drain it and discovered a growth coming through the posterior wall of the sinus from the dura, in the region of the crista galli. Growth was pushed back into the cranial cavity and the incision closed. Following this surgery the choked disks cleared up. Three months before admission a swelling in the region of the healed incision developed.

Physical and Neurological Findings: Fluctuant swelling on right side of forehead, extending down to the supraorbital ridge. Proptosis of right eye. Poor convergence. Sense of smell intact. Right corneal reflex absent. Loss of pain sensation in right eyelid.

Roentgen Findings: Extensive thickening on right side of lesser wing of the sphenoid, roof of the orbit, and inner table of the frontal bone up as far as the coronal suture. Anterior clinoid processes blunted; no dorsum sellae visible. Large, fuzzy density arising from the right lesser wing of the sphenoid, extending back into the cranial cavity and projecting over the sella. Ethmoid sinus on right side clouded. Falx calcified and displaced to left.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Meningioma of right sphenoidal ridge.

Operative Procedure: Patient was followed until November 1940 without operation because it was feared a meningitis might develop from the infected frontal sinus. On 11-26-40 a right temporofrontal craniotomy was performed with exposure and partial removal of a large meningioma springing from the right sphenoidal ridge, involving the anterior and middle cranial fossae. The medial border was not removed, for fear of running into the internal carotid artery.

Postoperative Course: The wound became infected and osteomyelitis developed in the bone flap. Patient refused further surgery. Last heard from in July 1947.

Pathologic Findings: Tumor weighed 97 gm.; measured 9 cm. in diameter and 7 cm. in thickness. Meningioma Type I, variant 4.

Survival Period: 7 years known survival.

CASE 17: A. McC., age 49, white female, admitted 5-26-38.

Chronology of Symptoms: Twelve years previously patient slipped and struck her left forehead on a corner of a dresser. A lump soon appeared over the left eye. Following x-ray therapy, the lump diminished in size, and pain and eye symptoms subsided. Well for five years. Seven years prior to admission pain again occurred in the left face and

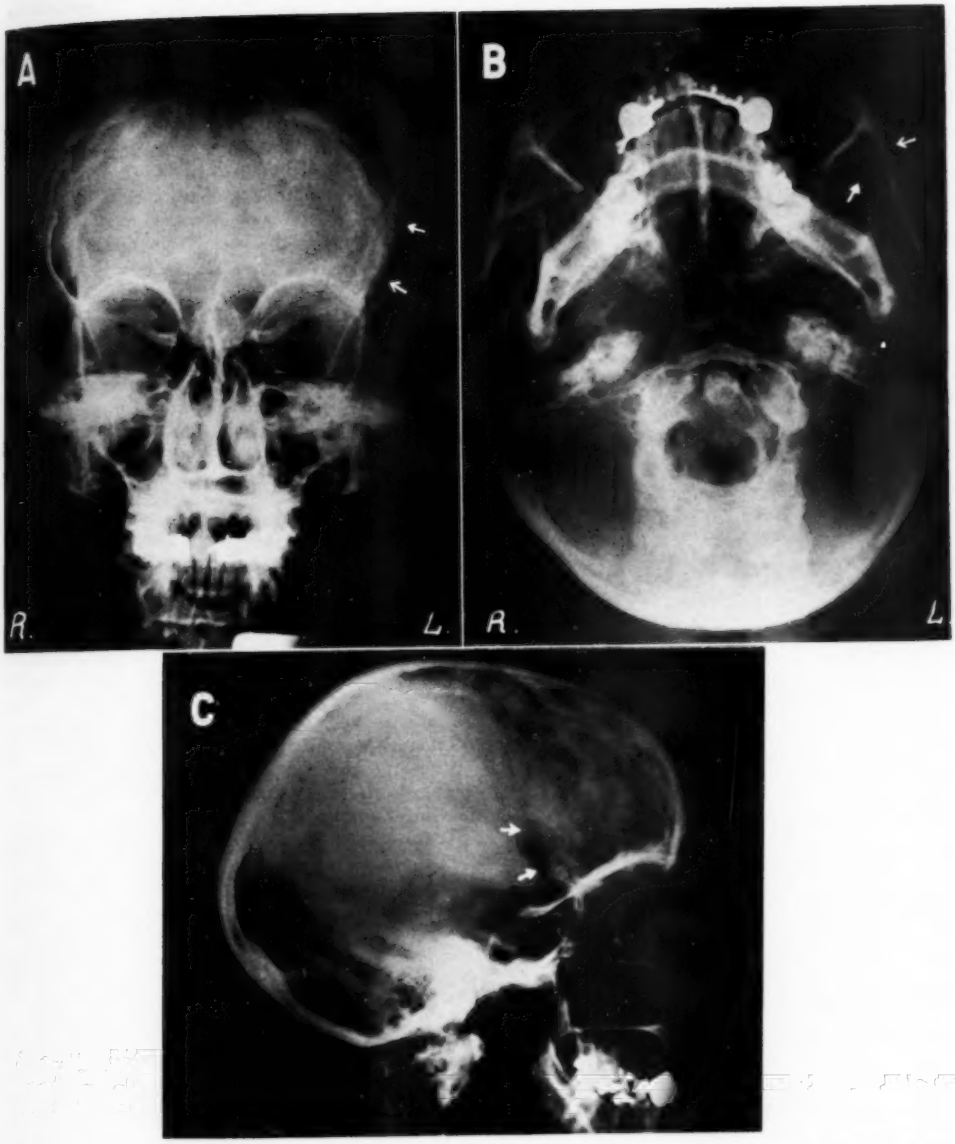

Fig. 17. Case 24: A 28-year-old white female gave an eighteen-month history of a swelling under the left ear, associated with tinnitus. Twelve months prior to admission to the hospital headache developed over the left eye and vision became blurred in both eyes. One week prior to hospital admission the spinal fluid pressure was 400 mm. of water. Examination showed bilateral mydriasis with gross contraction of visual fields with almost total loss of vision in the left eye and bilateral papilledema of 3 to 4 diopters. Operation revealed a *global* sphenoidal ridge meningioma on the left side, 5 cm. in diameter.

A. Postero-anterior view showing an area of mottled bone in the region of the lateral border of the sphenoidal ridge above the left orbit (arrows). The inferior portion of the greater wing of the sphenoid appears washed out on the left side.

B. Base view showing an area of increased density which has partially destroyed the curvilinear line which represents the anterior-superior border of the greater wing of the sphenoid (arrows).

C. Left lateral view showing an area of mottled bone due to increased vascularity in the pterional region (arrows).

the lump returned on left forehead. It grew gradually, and the eye became proptosed. Vision gradually diminished in the left eye, and more recently in the right. During these seven years patient received x-ray therapy, with subsidence of pain and some regression of the lump. Once the radiation therapy was stopped, the symptoms returned. During the eighteen months prior to admission there was a marked increase of exophthalmos, with total blindness in the left eye.

Physical and Neurologic Findings: Large bony tumor in left frontoparietal-temporal region, covered by a telangiectatic scar. Marked exophthalmos of the left eye, with corneal keratitis and a cataract. Patient unable to concentrate, uninterested, reticent, and quiet. Blindness in left eye. Impaired hearing, left ear. Impaired sense of smell on left side. Bilateral papilledema.

Roentgen Findings: Huge area of bone destruction involving left orbit and left side of frontal and temporal bone. Lesion extends into the base and there is considerable thickening of the lesser and greater wings of the sphenoid on the left side.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Meningioma of the left sphenoidal ridge.

Operative Procedure (6-1-38): Left frontotemporal bone flap turned down, with exposure and partial removal of a tumor involving the left frontal bone, together with the orbital plate and base of the skull in the temporal region.

Postoperative Course: Patient died 9-19-38. Autopsy revealed metastases in the lungs and in the left frontal fossa, with direct extension through the optic foramen.

Pathologic Findings: Total weight of tissue removed at operation, 250 gm. The bone measured $10 \times 6 \times 3$ cm. Attached to it were the galea aponeurotica and a mass of tumor tissue. One piece of tumor measured $10 \times 8 \times 3$ cm. Meningioma Type VI, variant 1. The metastases in the lungs resembled the tumor tissue removed at operation.

Survival Period: 3 months.

CASE 18: P. P., age 25, white female, admitted 6-14-38.

Chronology of Symptoms: Convulsion two years before admission, followed by a period of unconsciousness. No jacksonian convulsions. Total of about ten convulsions in two years. During last year, two to four attacks of dizziness daily, associated with a pounding in the right ear. Personality changes in the last few months.

Physical and Neurologic Findings: Bilateral early papilledema. Left central facial weakness. Left palpebral fissure wider than right. Hyperactive reflexes on left.

Roentgen Findings: Evidence of thinning and erosion of dorsum sellae from the top; displacement of the pineal to the left of the midline.

Ventriculographic Findings: Left lateral ventricle moderately dilated and slightly displaced to the left. Right lateral ventricle contains only a small amount of air and its lumen is almost obliterated. Obliteration of posterior portion of third ventricle; entire superior portion of third ventricle displaced to the left. Mass lesion in right parietotemporal region.

Encephalography: Not done.

Preoperative Diagnosis: Temporal lobe tumor on right side.

Operative Procedure (6-17-38): Right frontoparietal bone flap turned down, with exposure and removal of a meningioma arising from the greater wing of the sphenoid on the right side.

Postoperative Course: Wound healed promptly. Patient alive in August 1948, and neurologic findings negative.

Pathologic Findings: Weight of tumor 25 gm., $5 \times 3 \times 3$ cm. Meningioma Type I, variant 3.

Survival Period: 10 years. Living and well.

CASE 19: M. W., age 47, white female, admitted 8-31-38.

Chronology of Symptoms: Headaches began in April 1938. Prior to this, patient had experienced mental changes, with loss of memory and judgment. About June 1938 unsteady gait developed. 8-26-38, severe headache, vomiting, and coma. Decompression done on 8-28-38, with improvement.

Physical and Neurologic Findings: Slight exophthalmos of right eye. Rigidity of arms with cogwheel movement on left. Slight left facial weakness and weakness of tongue. Hyperactive reflexes and positive Babinski bilaterally.

Roentgen Findings: Slight distortion of anterior clinoid on right.

Ventriculographic Findings: Slight dilatation of left ventricle. Right lateral ventricle encroached upon and pushed up and toward the left. The appearance is that of a deep-seated mass lesion in the frontotemporal region.

Encephalography: Not done.

Preoperative Diagnosis: Mass lesion in frontotemporal region.

Operative Procedure (9-7-38): Right frontotemporal craniotomy, with exposure and removal of a tumor, 7 cm. in diameter, attached to the right greater wing of the sphenoidal ridge.

Postoperative Course: Alive on 10-12-44, with left-sided hemiplegia.

Pathologic Findings: Meningioma weighing 75 gm., Type II, variant 2.

Survival Period: 6 years, 1 month. Lost to follow-up.

CASE 20: M. L., age 50, white female, admitted 11-24-38.

Chronology of Symptoms: Shooting pains in back of head and neck of one year duration. For five months noticed vision of left eye failing. Deaf for many years following otitis media at age of sixteen.

Physical and Neurologic Findings: Bilateral

papilledema of 2 diopters. Suggestion of bilateral exophthalmos. Constriction of visual field of left eye, with cut in inferior nasal portion.

Roentgen Findings: Increased density of left sphenoidal ridge and erosion of one side of dorsum sellae. Pineal displaced posteriorly, downward and slightly to the right.

Ventriculographic Findings: Huge soft-tissue mass in left middle fossa displacing all structures to right side.

Encephalography: Not done.

Preoperative Diagnosis: Left sphenoidal meningioma.

Operative Procedure (11-29-38). Left frontoparietal craniotomy, with partial removal of a left sphenoidal ridge meningioma (inner third).

Postoperative Course: Fine condition and freedom from pain for about six years. In September 1948 patient became confused and unable to walk.

Pathologic Findings: Meningioma (psammoma) Type II, variant 3.

Survival Period: 10 years. Living in 1948.

CASE 21: J. S., age 47, white male, admitted 3-7-39.

Chronology of Symptoms: Struck in right orbit by a flying piece of steel in 1934. Two months later noticed right eye was becoming prominent. Prominence gradually increased and vision became blurred. Diplopia on looking to the right developed; also headaches, most noticeable on arising, subsiding half an hour later.

Physical and Neurologic Findings: Exophthalmos of right eye. Bruit heard over the globe. Right temporal vessels dilated and tortuous. Conjunctivae injected. Convergence of right eye diminished.

Roentgen Findings: Pineal shifted to left.

Ventriculography: Not done.

Encephalographic Findings: No air entered the ventricular system, but the basal cisterna filled and appeared normal.

Preoperative Diagnosis: Orbital tumor.

Operative Procedure: Intraorbital tumor removed, and diagnosed as a fibroma. Patient did well for a while, but on 12-16-39 a right frontoparietal craniotomy was performed, with partial removal of a right sphenoidal ridge meningioma. The tumor was attached all along the ridge.

Postoperative Course: Death 3-16-40.

Pathologic Findings: Tumor removed weighed 110 gm. Meningioma Type IV, variant 1.

Survival Period: 3 months.

CASE 22: W. J., age 20, white male, admitted 10-24-39.

Chronology of Symptoms: Headaches for many years. Fourteen months prior to admission, sudden loss of sight of left eye. Two months prior to admission headaches became worse and left-sided, and paresthesia of left side of face was noticed.

Physical and Neurologic Findings: Bilateral optic atrophy more marked on left. Ptosis and complete loss of vision in left eye. Corneal anesthesia of left eye. Sensory disturbance of left side of face. Tongue deviates to right. Hyperreflexia of right arm and leg.

Roentgen Findings: Enlargement and deformity

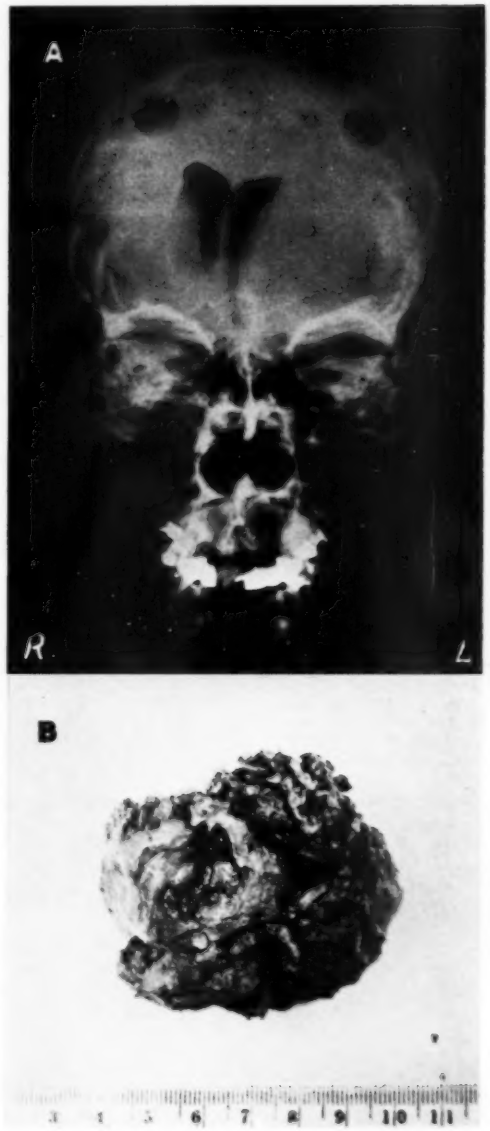

Fig. 18. Case 24, shown also in Fig. 17.

A. Postero-anterior ventriculogram showing displacement of both lateral and third ventricles to the right.

B. Photograph of the *global* meningioma, Type III, variant 2, removed from the left sphenoidal ridge. (Courtesy Armed Forces Institute of Pathology. Accession number 218242–4.)

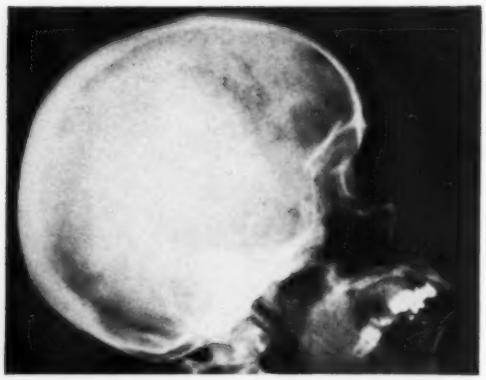

Fig. 19. A 42-year-old white female whose left eye had been protruding for six months had noticed blurred vision for two months. She had also lost 50 pounds in the two years prior to her admission to the hospital. She gave a history of having had a radical mastectomy six years previously. Examination showed marked exophthalmos of the left eye, which was nearly blind, anosmia, some anesthesia of the left side of the face, and protrusion of the tongue to the right. The roentgenogram shows an extensive lesion on the left side involving the frontal and anterior portion of the parietal bone. The dorsum of the sella is intact, but the entire anterior half of the sella, including the body of the sphenoid and lesser wings of the sphenoid, is missing. This was thought to be a meningioma arising from the left sphenoidal ridge. The patient was operated on and a huge metastatic carcinoma was found and partially removed. The bone of the vault was diagnosed as osteitis fibrosa. The patient died soon after operation.

of sella turcica. Destruction of left greater wing of the sphenoid and optic foramen.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Left-sided mass in region of sphenoidal ridge.

Operative Procedure (11-9-39): Left frontoparietal bone flap, with biopsy of tumor, which lay on the left greater wing of the sphenoidal ridge. Because of bleeding, tumor could not be removed. Second unsuccessful attempt 5-4-40.

Postoperative Course: Patient died 7-17-43.

Pathologic Findings: Meningioma Type IV, variant 1.

Survival Period: 3 years, 8 months.

CASE 23: J. P., age 62, white male, admitted 7-10-42. (Fig. 10)

Chronology of Symptoms: Five weeks before admission, loss of sense of equilibrium, nausea, loss of memory, and headache.

Physical and Neurologic Findings: Papilledema of both eyes, worse on right. Romberg positive, with fall to right. Slow response to questions. Slow broad-based gait. Air and bone conduction better on right. Electroencephalography indicated a lesion of the right frontal lobe. Slight left facial weakness.

Roentgen Findings: Negative.

Ventriculographic Findings: Right lateral ventricle did not fill. Left lateral and third ventricle displaced to left and somewhat dilated. Posterior portion of third ventricle did not show. Findings seemed to indicate a lesion in the inferior portion of the right frontal or temporal lobe.

Encephalography: Not done.

Preoperative Diagnosis: Right frontal or temporal lobe tumor.

Operative Procedure (7-11-42): Right frontoparietal craniotomy, with removal of a right sphenoidal ridge meningioma.

Postoperative Course: Patient sent home from hospital in poor condition. Died 9-1-42 of pulmonary condition.

Pathologic Findings: Meningioma Type II, variant 2.

Survival Period: 2 months.

CASE 24: H. S., age 28, white female, admitted 2-7-42. (Figs. 17 and 18)

Chronology of Symptoms: Swelling under left ear and tinnitus of eighteen months duration. Blurred vision and headaches over left eye, twelve months. Vision gradually became very poor in left eye and supraorbital headaches on left side became very severe.

Physical and Neurologic Findings: Bilateral mydriasis; gross contraction of visual fields, with almost total loss of vision in left eye. Bilateral papilledema of 3 to 4 diopters. Exaggerated deep reflexes, with a positive Babinski on left.

Roentgen Findings: Area of mottling and increased density in region of sphenoidal ridge, above the left orbit. Lateral view shows this to be in pterional region. Base view shows increased density in greater wing of sphenoid on left.

Ventriculographic Findings: Lateral ventricles and third ventricle of normal size, but displaced to the right, with most pressure on lateral wall of anterior horn of left lateral ventricle.

Encephalography: Not done.

Preoperative Diagnosis: Mass lesion in the right temporofrontal region.

Operative Procedure (12-7-42): Left frontotemporoparietal bone flap, with exposure and removal of a large, fairly well encapsulated tumor springing from outer edge of left sphenoidal ridge.

Postoperative Course: Bone flap removed, 12-26-42, because of edema and need for decompression. Vision returned in right eye and patient became free of symptoms.

Pathologic Findings: Poorly encapsulated tumor, 5 cm. in diameter, adherent to dura. Meningioma Type III, variant 2.

Survival Period: 6 years, 2 months. Still living.

CASE 25: B. G., age 37, white female, admitted 4-25-45.

Chronology of Symptoms: Nine-year history of headache. November 1944, blurring of vision in both eyes developed, worse on right.

Physical and Neurologic Findings: Bilateral papilledema with atrophy and exudate. Patient sees only light with right eye and reads small print with left. Deep reflexes absent in lower extremities. ? Left central facial nerve palsy. Papilledema: $2^1/_2$ diopters, right eye; 3 diopters left eye.

Roentgen Findings: Demineralization of posterior clinoids, dorsum sellae, and floor of sella. Pineal gland shifted 6 mm. to left of midline.

Ventriculographic Findings (5-10-45): Moderate shift of ventricular system toward left and a deformity of right lateral ventricle, indicating presence of mass lesion in right hemisphere, probably in frontoparietal or frontotemporal region.

Encephalography: Not done.

Preoperative Diagnosis: Mass lesion in frontotemporal region.

Operative Procedure (5-10-45): Right frontotemporal craniotomy, with exposure and partial removal of a meningioma *en plaque* springing from outer edge of sphenoidal ridge. No attempt made to remove sphenoidal ridge, but dura removed and the base coagulated.

Postoperative Course: Patient completely blind in right eye except for light perception. Has had right frontal headaches ever since operation, with retro-orbital pain and pain in right temple. Roentgenography shows increased density of right sphenoid bone and supra-orbital portion of right frontal bone. No further surgery performed to date.

Pathologic Findings: Mass of tissue, 5.5 × 4 × 3.5 cm., weighing 40 gm., consisting of both tumor and dura. Meningioma Type I, variant 1.

Survival Period: 2 years, 8 months. Living 1-14-48.

CASE 26: A. B., age 23, white female, admitted 5-5-45.

Chronology of Symptoms: Diplopia in February 1941. In 1943, trouble with vision, particularly on looking to right. At same time patient began to gain weight. May 1944, complete amenorrhea developed. In last few months considerable impairment of vision, particularly in right eye.

Physical and Neurologic Findings: Primary optic atrophy, both eyes: complete on right; loss of temporal field in left eye.

Roentgen Findings: No evidence of increased intracranial pressure. Pituitary fossa not enlarged but dorsum sellae eroded from above and downward; appearance typical of suprasellar lesion.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma, based on clinical rather than radiological opinion.

Operative Procedure (5-5-45): Right frontotemporal bone flap, with exposure and partial removal of a meningioma involving the inner end of sphenoidal ridge and probably extending over to left side. Impossible to remove very much of tumor because it lay so far medially in region of internal carotid.

Postoperative Course: May 1947, patient alive but doing very poorly.

Pathologic Findings: Six pieces of granular tumor, each about 1 cm. in diameter. Meningioma Type I, variant 2.

Survival Period: 2 years when last heard from in May 1947.

CASE 27: N. R., age 43, white female, admitted 5-26-45.

Chronology of Symptoms: Since 1930 had trouble controlling her right eye, as it tended to waiver. In 1941 began having dull headaches in region of right eye and temple, and about the same time noted a bulging of right temple. In 1943 proptosis of right eye developed. No diplopia, blurring of vision, or anosmia.

Physical and Neurologic Findings: Palpable swelling in right temporal region. Slight proptosis of right eye but visual fields normal.

Roentgen Findings: Right-sided hyperostosis, size of a golf ball, involving roof and lateral wall of orbit, and greater and lesser wings of the sphenoid from pterional region to their inner third.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (5-29-45): Right frontal craniotomy, with removal of roof and lateral wall of orbit and a large bony tumor involving the lesser and greater wings of the sphenoid.

Postoperative Course: 6-27-49, patient very well.

Pathologic Findings: Numerous pieces of dense bone with attached dura. Section of dura showed it to be infiltrated by a meningioma of whorl format, Type II, variant 1.

Survival Period: 4 years, 1 month. Living and well.

CASE 28: A. H., age 57, white female, admitted 7-6-45. (Fig. 20)

Chronology of Symptoms: Patient committed to a mental institution in March 1943, because of a paranoid state. At that time there was an exophthalmos of right eye and roentgenogram showed a destruction of roof of right orbit on nasal side and opacity of ethmoid sinuses on both sides. September 1944, mass in right temporal region noticed. Soon after this, patient lost vision of right eye and eyelids became edematous. Mass gradually extended from mid-frontal to right upper parietal region.

Physical and Neurologic Findings: Head greatly deformed and asymmetrical, with a huge bulging of frontal, parietal, and malar structures of skull. Mass was bluish and cystic and pushed the eye downward and inward. Left eyeground showed enlarged veins and a fuzzy disk. Right eye not visualized. Reflexes generally decreased.

Roentgen Findings: Large soft-tissue mass arising in region of right frontal bone. Erosion of lateral wall of orbit, adjacent frontal bone, greater wing of

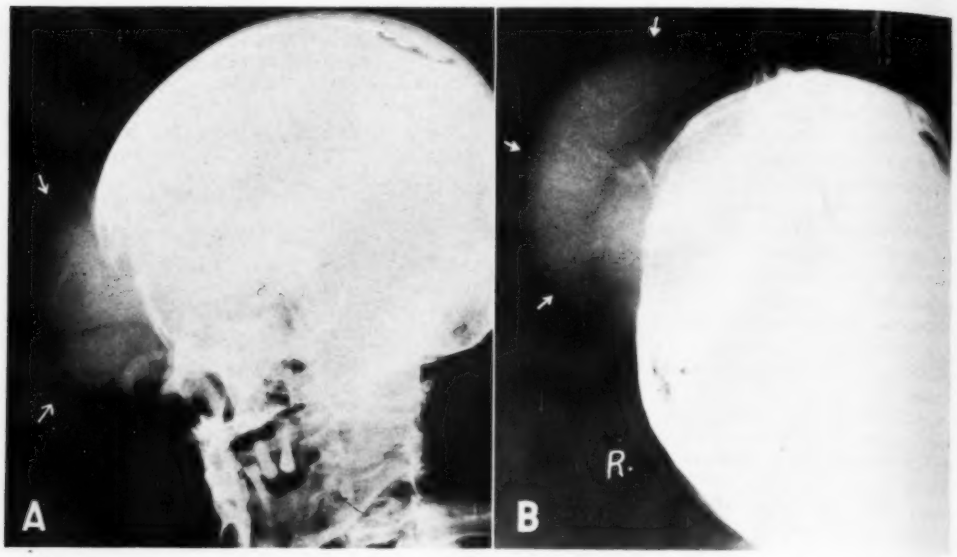

Fig. 20. Case 28: A 57-year-old white female had been committed to a mental institution because of a paranoid state two years prior to hospital admission. At that time there was an exophthalmos of the right eye. After one year in the mental institution a mass appeared on the right side in the frontotemporoparietal region. Soon after this the patient lost the sight of her right eye. Examination showed a huge bluish mass bulging from the right frontotemporoparietal region. The right eye was pushed far out on the surface of the mass. There was papilledema of the left eye. The patient was not operated upon, and ten days later she died of a cerebrovascular accident. Autopsy showed a soft nodular tumor involving the entire right anterior cranial fossa, orbit, and sphenoidal ridge. The tumor was diagnosed as a meningioma Type III, variant 4.

A. Tangential view showing the bone erosion and the bony spicules within the tumor mass.

B. Base view showing the large soft-tissue mass with bony spicules extending outward from the right pterional region.

sphenoidal bone, and to a less extent the lesser wing of sphenoidal bone. Perpendicular striations of bone visible in tangential and Hirtz views.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Osteogenic sarcoma.

Course: Patient was not fit for surgery. Ten days after admission she went into auricular fibrillation and died of a cerebrovascular accident.

Necropsy Findings: Entire right anterior fossa, orbit, and sphenoidal ridge replaced by a soft nodular tumor. Meningioma Type III, variant 4.

Survival Period: Died in hospital.

CASE 29: F. P., age 21, white male, admitted 7-26-45.

Chronology of Symptoms: Bulging of right eye noticed in 1940, progressing within a week to the degree present on admission. No disturbance of vision.

Objective Findings: Exophthalmos of right eye, with eye pushed downward.

Roentgen Findings: Dense radiopacity measuring 3 cm. in diameter overlying lateral margin of sphenoidal ridge.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (8-14-45): Right frontotemporal bone flap, with exposure and partial removal of an extradural meningioma from right sphenoidal ridge and roof of right orbit, as well as along the right olfactory groove.

Postoperative Course: Favorable.

Pathologic Findings: Tumor, removed piecemeal, weighed 50 gm. Meningioma (osseous type) Type VII.

Survival Period: Patient not traced.

CASE 30: M. N., age 47, white female, admitted 9-18-45.

Chronology of Symptoms: Two years prior to admission patient fell and hit the back of her head, after which there were two episodes of inflammation and soreness of the right eye, lasting about a month. Four months prior to admission patient noticed right eye was beginning to bulge; one month prior to admission blurring of vision and diplopia. Constant dull ache in right occipital region ever since falling.

Physical and Neurologic Findings: Exophthalmos of right eye. Tendency to fall to left. Visual fields normal. Hard bony mass in right temple region.

Roentgen Findings: Diffuse area of increased density in region of greater and lesser wings of right sphenoidal bone, with encroachment upon the sphenoidal fissure. Optic foramina normal.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (9-28-45): Right frontal craniotomy, with exposure and removal of a bony tumor involving roof of orbit and entire sphenoidal ridge. The actual meningioma *en plaque* was not removed because, by accident, the ethmoid sinus was opened. 10-16-45, same bone flap laid back and an intradural tumor lying along the sphenoidal ridge removed.

Postoperative Course: Recovery uneventful. However, a chronic area of granulation tissue developed in the region of the wound, which required pinch grafts on 1-27-46. After this the wound healed.

Pathologic Findings: Thickened dura 4.5 × 2.5 × 1.5 cm., infiltrated by meningioma Type III, variant 1.

Survival Period: 15 months. Alive and well when last heard from, January 1947.

CASE 31: C. T., age 3 months, white female, admitted 5-8-46.

Chronology of Symptoms: At birth it was noticed that the patient's eye protruded, with a slight swelling about the right temple. At five weeks, due to frequent vomiting, the weight was less than at birth. At the time of admission the right temple was gradually increasing in size.

Physical and Neurologic Findings: Moderate proptosis of right eye, with palpable swelling behind the upper lid. Moderate soft fluctant swelling of right temporal region. Right eyelids edematous and red. Babinski sign positive bilaterally.

Roentgen Findings: Right orbital cavity larger and more dense than left.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right.

Operative Procedure (5-22-46): Right transfrontal craniotomy, with exposure and removal of orbital roof and possible tumor of orbit with a meningioma lying along the outer edge of the sphenoidal ridge and temporal bone.

Postoperative Course: Proptosis of right eye continued; on 1-10-47 the orbit was further explored and more tumor found. On 2-21-47 the right eye was enucleated.

Pathologic Findings: Meningioma Type II, variant 2.

Survival Period: Two years plus. Still alive and apparently well.

CASE 32: H. C., age 42, white female, admitted 6-25-46. (Figs. 6–8)

Fig. 20C. Photograph showing large defect on the inferior surface of the right frontal lobe extending back to the temporal lobe. The entire region around the right lateral fissure appears soft and friable. (This and Fig. 20A and B are by courtesy of the Armed Forces Institute of Pathology, Accession number 218229–1 to 3.)

Chronology of Symptoms: Chronic draining right ear for thirty-seven years. Right-sided earache for three months, with pain in right side of head and failing vision in right eye with diplopia.

Physical and Neurologic Findings: Papilledema: 3 diopters on right and 1 diopter on left. Left central facial weakness. Questionable left hemiparesis. Increased deep reflexes on left. Babinski sign bilaterally positive. Questionable clonus of left ankle. Definite torpor and mental deficiency.

Roentgen Findings: Right mastoid diffusely clouded and sclerotic.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Acute mastoiditis, with abscess on right side.

Operative Procedure: 6-26-46, suspected abscess needled but no abscess found; 6-27-46, right mastoidectomy, but no abscess found.

Postoperative Course: Patient scheduled for air study and craniotomy on 6-29-46 but died eight hours earlier.

Pathologic Findings: Well encapsulated tumor, 5 × 5 × 4 cm., attached to right sphenoidal ridge. Meningioma Type II, variant 5.

Survival Period: Two days after mastoidectomy.

CASE 33: G. S., age 40, white female, admitted 7-18-46.

Chronology of Symptoms: In September 1944 patient became tired, drowsy, dizzy, and finally unconscious for a short period. Five months later she had another attack. Decrease of visual acuity of right eye with pain over eye for two years.

Physical and Neurologic Findings: Exophthalmos of right eye. Optic atrophy of right disk, with blurring of nasal border of left disk. Superior nasal quadrant right field destroyed; left field normal. Weakness of right 3rd nerve. Decreased corneal reflex on right. Cerebrospinal fluid pressure, 275 mm. of water.

Roentgen Findings: Sclerosis in region of right posterior ethmoid surrounding the optic foramen. Increased density of the greater and lesser sphenoidal wings on right. Complete obliteration of right sphenoidal sinus.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (7-23-46): Right frontoparietal craniotomy, with partial removal of a meningioma *en plaque* arising from inner third of the sphenoidal ridge and extending back into the middle fossa.

Postoperative Course: Wound treated with no complications, but as bone was involved, the tumor may recur.

Pathologic Findings: Meningioma Type II, Variant 3.

Survival Period: Patient not traced.

CASE 34: M. L., age 42, white female, admitted 10-3-46.

Chronology of Symptoms: Bulging of left eye noticed five years prior to admission, increasing gradually until ten months prior to admission. Patient complained that foods smelled badly to her, especially hot foods.

Physical and Neurologic Findings: Exophthalmos of left eye; slight pallor of disk. Decreased auditory acuity on right.

Roentgen Findings: Increased calcification of left sphenoidal ridge.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on left side.

Operative Procedure (10-8-46): Left frontal craniotomy, with partial removal of a meningioma *en plaque* from left sphenoidal ridge. Decompression of left orbit also done.

Postoperative Course: Patient did well postoperatively but the tumor invaded the bone and this was not all removed. Patient free of symptoms when heard from in 1947.

Pathologic Findings: Four tiny pieces of tumor removed. Meningioma Type IV, variant 3.

Survival Period: Only 1 year follow-up. Living and well in 1947.

CASE 35: R. G., age 44, white female, admitted 1-6-47.

Chronology of Symptoms: Swelling of right upper eyelid noticed in 1943. A short time later right eye became very prominent and a swelling of right temporal region developed. In 1945, generalized headaches and numbness of hands and feet developed; in 1946, loss of sense of equilibrium and loss of fine movements of hands.

Physical and Neurologic Findings: Swelling of right temporal region. Exophthalmos of right eye; nystagmus on looking upward. Deviation of mouth to left and slight decrease of olfaction on left.

Roentgen Findings: Increased density of lesser and greater wings of right sphenoid and of roof of right orbit. Superior orbital fissure on right narrowed by hyperostosis of sphenoid.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (1-10-47): Right frontotemporal bone flap, with exposure and possibly complete removal of meningioma involving sphenoidal ridge and orbital roof.

Postoperative Course: Rapid recovery. As of November 1948 patient had no neurologic findings and felt well.

Pathologic Findings: Meningioma Type II, variant 1.

Survival Period: 1 year, 11 months. Living, well.

CASE 36: A. C., age 49, white male, admitted 2-27-47.

Chronology of Symptoms: Sudden episode of unconsciousness late in 1944 lasting 30 minutes. Second attack one year later; third six months prior to admission, with incontinence of urine; 4th three weeks prior to admission, with biting of tongue. Severe occipital headaches since 1944, which radiate to frontal region, chiefly on right.

Physical and Neurologic Findings: Bilateral hyposmia. Slight left central facial paralysis. Bilateral papilledema of 3 to 4 diopters with exudates and hemorrhages around the disks. Visual fields normal.

Roentgen Findings: Dorsum sellae appears demineralized and irregular. Floor of right middle fossa more dense than left. Lateral portion of right sphenoidal ridge slightly irregular.

Ventriculographic Findings: Extensive shift of ventricular system to the right. Anterior horn, the body, and temporal horn are shifted.

Encephalography: Not done.

Preoperative Diagnosis: Right frontotemporal tumor, possibly a sphenoidal ridge meningioma.

Operative Procedure (3-6-47): Right frontotemporoparietal bone flap, with exposure and complete

removal of meningioma springing from sphenoidal ridge and involving right sylvian fissure. Necessary to clip large artery, probably a major branch of middle cerebral.

Postoperative Course: Good recovery, but with a left central hemiparesis involving the face and arm on the left. On 4-2-49 most of the hemiparesis had cleared and patient was working as a carpenter again.

Pathologic Findings: Meningioma Type II, variant 3; 68 gm.; 7.5 cm. long; 3.5 cm. in diameter.

Survival Period: 2 years, 2 months. Alive, well.

CASE 37: I. G., age 48, white male, admitted 7-10-47.

Chronology of Symptoms: Swelling in right temporal region noticed one year prior to admission; gradually grew to size of half an egg. At same time frontal headaches developed, becoming more severe in the last four or five months. Day before admission patient vomited twice. For last year noticed right eye was bulging.

Physical and Neurologic Findings: Hard, tender swelling about the size of a small egg in right frontotemporal region. Exophthalmos of right eye. Papilledema on right. Positive Babinski on left.

Roentgen Findings: Erosion of lesser and greater wings of sphenoid on right. Pineal shifted, displaced to left. Area of increased density in right temporoparietal region of vault, just above pterion.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on right side.

Operative Procedure (7-12-47): Right frontotemporoparietal bone flap, with exposure and partial removal of a meningioma involving the sphenoidal ridge above the orbit. Tumor invaded the bone and passed through into the temporal muscle.

Postoperative Course: Good recovery; return to former work.

Pathologic Findings: 79 gm. multilobular meningioma Type II, variant 2, 8 cm. in diameter and 2 cm. thick.

Survival Period: One year. Still living when last heard from in May 1948.

CASE 38: J. B., age 54, white male, admitted 9-22-47.

Chronology of Symptoms: Onset of diplopia and gradual blurring of vision, especially right field of vision, noticed six week before admission. Some loss of memory for two or three months.

Physical and Neurologic Findings: Bilateral 6th nerve weakness. Lower central facial paresis on right. Partial anosmia. Hesitant speech and forgetfulness for words. Three to 4 diopters papilledema bilaterally.

Roentgen Findings: Pituitary fossa enlarged and dorsum decalcified as result of increased intracranial pressure. Pineal displaced to the right, posteriorly, and inferiorly.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Glioma on left side.

Operative Procedure (9-27-47): Left frontotemporoparietal bone flap with exposure and removal of a large meningioma springing from outer edge of sphenoidal ridge.

Postoperative Course: Patient did well postoperatively. Returned to former work. Vision cleared up; no neurologic findings remain.

Pathologic Findings: Large tumor weighing 109 gm., 6.5 cm. in diameter and 3.5 cm. in depth. Meningioma Type I, variant 3.

Survival Period: 1 year, 8 months. Living, well.

CASE 39: S. Y., age 54, white female, admitted 11-6-47.

Chronology of Symptoms: Three years prior to admission patient suddenly felt a "crawling sensation over entire body" and then fell to the floor. These "spells" recurred several times in the next twelve months; last spell August 1945. No convulsions or loss of consciousness during these attacks. For two months prior to admission vision in right eye getting cloudy. Frontal headaches for a few months.

Physical and Neurologic Findings: Papilledema, 2 diopters, right disk; temporal pallor of left disk.

Roentgen Findings: Destruction of dorsum sellae and partial destruction of left anterior clinoid process. Increased density of left greater and lesser wings of sphenoid.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on left side.

Operative Procedure (11-13-47): Left frontotemporal craniotomy, with almost complete removal of sphenoidal ridge meningioma. All tumor removed except for a little surrounding the optic nerve and carotid artery.

Postoperative Course: Patient ambulatory fifth day. Alive and free of any neurologic changes.

Pathologic Findings: Tumor removed piecemeal; 50 gm. Meningioma Type II, variant 1.

Survival Period: 1 year, 5 months.

CASE 40: A. L. age 12, white female, admitted 1-19-48.

Chronology of Symptoms: Double vision first noticed one year prior to admission. June 1947, left eye became proptosed. October 1947, headaches began.

Physical and Neurologic Findings: Exophthalmos of left eye. Paresis of left 3rd cranial nerve, with loss of action of levator, superior rectus, inferior oblique, and inferior rectus Early papilledema of left disk of 1 diopter.

Roentgen Findings: Sella top-normal in size. Erosion of left anterior clinoid along inferior surface.

Ventriculography: Not done.

Encephalographic Findings: (2-6-48): Slight asymmetry of the ventricular system, with occipital horn on left side elevated.

Preoperative Diagnosis: First preoperative diagnosis was intraorbital tumor; diagnosis after encephalography was parasellar lesion on left.

Operative Procedure (1-27-48): Left orbit explored and nothing found. 2-11-48, left frontoparietal craniotomy with exposure and partial removal of a sphenoidal ridge meningioma attached to inner one-third of ridge and filling most of middle fossa on left.

Postoperative Course: Alive and well; complete 3rd nerve paralysis with ptosis developed. On 5-5-49 transplant operation done because of ptosis.

Pathologic Findings: Tumor removed piecemeal. Meningioma Type III, variant 1.

Survival Period: 16 months. Alive and well.

CASE 41: J. S., age 52, white male, admitted 11-5-48.

Chronology of Symptoms: Proptosis of left eye first noticed in January 1946, with possibly a little visual impairment. Roentgenograms elsewhere reported as negative. January 1947 further roentgen examination, reported as negative. Patient remained asymptomatic except for proptosis. On 10-1-48 lesion of left sphenoidal ridge finally diagnosed.

Physical and Neurologic Findings: Proptosis and protrusion of left eye. Vision in left eye 20/30 with correction and of right eye 20/20 with correction

Roentgen Findings: Thickening of entire left sphenoidal bone and roof of left orbit. Pituitary fossa not altered. Pineal gland not displaced.

Ventriculography and Encephalography: Not done.

Preoperative Diagnosis: Sphenoidal ridge meningioma on left side.

Operative Procedure (11-9-48): Left frontotemporal bone flap, with exposure and removal of tumor involving sphenoidal wing and roof of left orbit and dura along the posterior edge of the sphenoidal ridge.

Postoperative Course: Left hospital on twentieth postoperative day. Low-grade infection in wound gradually cleared up with antibiotics.

Pathologic Findings: Dura measuring 4 × 5 cm. and 0.75 cm. thick, invaded by meningioma Type II, variant 4.

Survival Period: 7 months. Alive and well.

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REFERENCES

1. CUSHING, H., AND EISENHARDT, L.: Meningiomas: Their Classification, Regional Behaviour, Life History, and Surgical End Results. Springfield, Ill., Charles C Thomas, 1938.

2. WEED, L. H.: In Cytology and Cellular Pathology of the Nervous System, edited by W. PENFIELD, New York, Paul B. Hoeber, Inc., 1932.

3. GRINKER, R. R.: Neurology. Springfield, Ill., Charles C Thomas, 3rd. ed., 1943.

4. PENFIELD, W.: Cytology and Cellular Pathology of the Nervous System. New York, Paul B. Hoeber, Inc., 1932.

5. PANCOAST, H. K., PENDERGRASS, E. P., AND SCHAEFFER, J. P.: The Head and Neck in Roentgen Diagnosis. Springfield, Ill., Charles C Thomas, 1940.

6. ARIENS KAPPERS, C. U.: Meninges in Lower Vertebrates Compared with Those in Mammals. Arch. Neurol. & Psychiat. **15:** 281–296, 1926.

7. HARVEY, S. C., AND BURR, H. S.: Development of the Meninges. Arch. Neurol. & Psychiat. **15:** 545–567, 1926.

8. HARVEY, S. C., BURR, H. S., AND VAN CAMPENHOUT, E.: Development of the Meninges; Further Experiments. Arch. Neurol. & Psychiat. **29:** 683–690, 1933.

9. LEARY, T., AND EDWARDS, E. A.: The Subdural Space and Its Linings. Arch. Neurol. & Psychiat. **30:** 691–701, 1933.

10. GLOBUS, J. H.: The Meningiomas, Their Origin, Divergence in Structure, and Relationship to Contiguous Tissues ... with a Suggestion of a Simplified Classification of Meningeal Neoplasms. Proc. A. Research Nerv. & Ment. Dis. (1935) **16:** 210–265, 1937.

11. GLOBUS, J. H., LEVIN, S., AND SHEPS, J. G.: Primary Sarcomatous Meningioma (Primary Sarcoma of the Brain). J. Neuropath. & Exper. Neurol. **3:** 311–343, 1944.

12. TURNER, O. A., CRAIG, W. M., AND KERNOHAN, J. W.: Malignant Meningiomas: Clinical and Pathologic Study. Surgery **11:** 81–100, 1942.

13. HAYMAKER, W.: Personal communication, 1948.

14. KAZAN, A. T., WELLER, D., AND GOMÉZ JARAMILLO, J.: Intracranial Meningiomas. J. Mt. Sinai Hosp. **11:** 105–128, 169–184, 1944.

15. CUSHING, H.: Cranial Hyperostoses Produced by Meningeal Endotheliomas. Arch. Neurol. & Psychiat. **8:** 139–154, 1922.

16. PHEMISTER, D. B.: Nature of Cranial Hyperostosis Overlying Endothelioma of the Meninges. Arch. Surg. **6:** 554–572, 1923.

17. KOLODNY, A.: Cranial Changes Associated with Meningioma, "Dural Endothelioma." Surg., Gynec. & Obst. **48:** 231–235, 1929.

18. ROWBOTHAM, G. F.: Hyperostoses in Relation with the Meningiomas. Brit. J. Surg. **26:** 593–623, 1938–39.

19. GRANT, F. C.: Intracranial Meningiomas, Surgical Results. Surg., Gynec. & Obst. **85:** 419–431, 1947.

SUMARIO

El Diagnóstico Roentgenológico de los Meningiomas de la Cresta Esfenoidal

El repaso de 41 meningiomas comprobados de la cresta esfenoidal demuestra que el diagnóstico fué establecido acertadamente por los hallazgos roentgenológicos en 35 casos (85.4 por ciento). Una vez realizada la extirpación cruenta total, puede ofrecerse un buen pronóstico a estos enfermos. En esta serie, transcurrió un período medio de tres años desde la iniciación de los síntomas hasta el ingreso en el hospital. Hay que evitar, si es posible, esa larga demora, por ser más fácil resecar las lesiones tempranas.

Esta reseña es para familiarizar a los radiólogos y a otros interesados con la típica semiología y hallazgos roentgenológicos de dicho tumor. La presencia de exoftalmía unilateral, trastornos visuales y edema en la región temporal debe hacer pensar siempre en la posibilidad de meningioma de la cresta esfenoidal. Si esa característica sintomatología se asocia a hiperostosis de las alas menores o mayores del esfenoides, cabe hacer por lo general un diagnóstico positivo.

Los signos roentgenológicos de un meningioma del tercio interno de la cresta esfenoidal consisten en hiperostosis o mayor espesor de la clinoides anterior ipsilateral, el conducto óptico y la porción interna del ala menor del esfenoides, lo cual se observa mejor en una vista posteroanterior y del canal óptico. Sino puede demostrarse la presencia de la lesión por las alteraciones óseas reveladas por las radiografías corrientes, la indicarán abiertamente las anomalías mostradas por la encefalografía, la ventriculografía o la ventriculoencefalografía.

Los tumores del medio de la cresta pueden revelar hiperostosis o absorción ósea de las alas menores y mayores del esfenoides. Una vista del seno frontal (sin diafragma de Bucky) resulta útil para mostrar las alteraciones de las crestas esfenoidales observadas a través de las órbitas.

Los tumores de la cresta esfenoidal, pterionales *en plaque*, provocan hiperostosis de las alas mayores y menores del esfenoides y en algunos casos, de la placa orbitaria del frontal, la porción escamosa del temporal y la región pterional. En las radiografías posteroanteriores, con las imágenes de las alas mayores y menores del esfenoides proyectadas en la órbita, resulta patognomónico su aspecto ebúrneo, contrastado con el del lado opuesto. Pueden también reconocerse las singulares alteraciones ebúrneas en la vista estereoscópica lateral, sobre todo en la región del pterión.

Los tumores globales del pterión producen hipervascularidad (múltiples surcos vasculares) en la región pterional, que se observa mejor en las radiografías estereoscópicas laterales.

DISCUSSION

(Papers by Young; Hodes, Pendergrass, and Young; Camp; Pendergrass, Hope, and Perryman)

Edwin Boldrey, M.D. (San Francisco): I regret the disappointment which I am sure many of you feel because of the absence of Dr. Naffziger, who is listed on the program as the one to open this discussion. However, I feel greatly honored at being asked by the officers of your Society to substitute for him. I consider myself most fortunate to have had the opportunity of reading and hearing the stimulating papers which have been presented this morning.

The mere fact of this Symposium is evidence of the major role played by roentgenology today as an aid in the diagnosis of tumors of the brain. The films which have been described were obtained because there was a possibility that the patient was afflicted by a tumor of the brain, and there was a good chance that plain roentgenograms would be of aid in establishing, or disestablishing, the presumptive diagnosis. It should be particularly emphasized that the reference is to

plain films. We are well acquainted with the added contribution of radiology when foreign materials are introduced into the system as differentiating media. I refer, of course, to the subarachnoid and intraventricular gases, the radiopaque oils and, more recently, radioactive isotopes.

We have all been interested in the displacement of physiologic intracranial calcification as discussed by Dr. Young. The pineal, of course, was the earliest of the normally calcified masses within the cranium to excite interest, and for the past quarter of a century we have used its displacement extensively as an early sign of the presence of brain tumor. I would like to endorse heartily and to substantiate Dr. Young's emphasis on the need of stereoscopic views in the anteroposterior and lateral projections in the study of pineal and other calcifications within the cranium. These help, not only in the establishment of the location of the pineal and the presence of the choroid plexus, but also in picking up bits of calcification within tumors themselves.

I would also like to comment on calcification in the falx, which was mentioned by Dr. Young. We often find that this calcification is unilateral. A mass of calcium or calcified deposit will be present along only one side of the falx, which will appear, therefore, to be displaced. By actual measurement of the center of the calcification, getting its distance from the lateral aspects of the cranium, one obtains apparent evidence of displacement of 2 or even 3 or 4 millimeters. This will be false in many instances, as the falx is a very firmly fixed structure, not permitting itself to be pushed aside as does the pineal. The ultimate limits of displacement of the falx are no more than 1 or 2 millimeters.

Dr. Hodes has brought up a matter of tremendous importance to us who are interested in tumors of the brain in his discussion of the tumors of the cerebellopontile angle. This problem is particularly stimulating because of the fact that, when these tumors are diagnosed early, they can be completely cured. Unfortunately, most of them come to our attention in the very late stages when they involve not only the 8th nerve, from which they generally arise, but also, as Dr. Hodes pointed out, the 7th, 5th, 3d, and sometimes the 9th, 10th, 11th, and 12th. Some may have involved the brain stem.

Traditionally these tumors can be dissected away from the brain stem and perhaps we should be able to do that under all circumstances. However, every neurosurgeon has found attachment to the basilar artery. There are also tumors which have become so intimately connected with the pia of the pons and brain stem that even on a histological preparation one is not able to determine where the tumor ends and the pia of the pons begins. Under such circumstances, we cannot hope successfully to dissect away the growth without seriously incapacitating the patient, or perhaps causing his death. These tumors must pass through an early stage, and if we can recognize them early, we can eradicate them completely. We are, of course, most interested in achieving this end and depend upon our radiological colleagues to help us.

Turning to the matter of the change in the sella turcica, we have also been interested in it as a clue to the duration of certain diseases. I am sure that all of us are most interested in the case presented by Dr. Camp, in which he has shown that changes developed in the sella turcica within thirty-seven days. It is a very important point to remember.

We have come to rely heavily on the changes in the sella turcica as indications of extra- or intrasellar disease. While the focal changes around the sella have interested us, we have not been ready to rely, as a rule, on the evidence in plain x-ray films alone. We often want to have encephalography or ventriculography, or, more recently, angiography. One cannot forget that aneurysms are a very potent source of focal distortion and destruction in the region of the sella turcica. A large percentage of the aneurysms which are clinically demonstrable occur in this area.

I would also like to mention, among the intrinsic tumors of this area, the chordoma. This tumor produces changes around the basisphenoid and basiocciput, primarily, but can grow into the floor of the sella and can also invade the dorsum. Chordomas are sometimes overlooked, or they may suggest the roentgenologic picture of an intrasellar lesion. Dr. Camp's point that we must get repeated postoperative studies must be emphasized. We forget that after we have altered the intracranial disease there are reverse changes, which may be reversed again by a recurrence of the neoplasm.

Tumors of the meningioma group are always interesting, because they carry a better prognosis than do most of the other major classes of intracranial neoplasms. The fact that Dr. Pendergrass' series included a colored patient is noteworthy, inasmuch as there has been some discussion in the literature of the past suggesting that this tumor was rare, if not unheard of, in the Negro race.

No one who has operated on meningiomas will deny that they are provokingly vascular. The vascularity has been well demonstrated, and we are learning more and more about it through the realm of angiography, in which the radiologist plays such a major role. The vascular supply is traditionally thought of as being from the external carotid but, by this method of study, it has been demonstrated that the internal carotid plays almost as great a role in the blood supply of these tumors as the external carotid. The venous blush, which was shown so well in the slide that

Dr. Pendergrass projected, is sometimes regarded as being pathognomonic of meningiomas. This assumption is probably unwarranted at this time. It is still early for us to rely too heavily on angiography in attempting probable pathological diagnosis of the lesion with which we are dealing.

The extracranial meningioma which Dr. Pendergrass presented was, in our experience, most extraordinary. It is even more interesting to know that there are 3 in his series.

A point with respect to the orbital meningiomas is the relationship of unilateral exophthalmos to this disease. We have seen several of these meningiomas invading the orbit and producing a unilateral exophthalmos. The diagnosis has been made on that basis. There is also a large series, which must not be forgotten, which produce no changes at all in the bone demonstrable either by x-rays or by actual inspection at the time of surgery or autopsy. Therefore, the positive diagnosis is most important. The negative diagnosis is only that, and cannot be used in deducing positive points.

During this discussion, it must have been obvious to all of you that the points made by these authors are just as important to the neurologist as they are to the radiologist. The contribution of roentgenology to the diagnosis of all types of disease of the nervous system has made it an integral part of most examinations of this system. This should in no wise be construed to depreciate the inestimable value of the clinical neurological examination. The information attained through our various senses will always form the ultimate basis of study of the patient. However, the roentgen ray gives us glimpses of anatomical changes beneath the skin and, in the study of tissue of the nervous system, we can ill afford to ignore any source of information which is valid and which offers probable benefits in excess of the possible danger to the patient. This applies particularly in the diagnostic evaluation of patients with intracranial neoplasm.

The papers of this session represent significant advances in the problem of recognizing the presence of brain tumor. However, it is sobering to realize that a considerable part of each of the four discussions pertains to the manifestation of late stages of neoplastic disease. The brain already will have suffered irreparable damage. The superimposition of further neurological complications will already have been commenced in many instances. We must diagnose brain tumors earlier. I believe that through our concerted efforts we will accomplish this end.

Kenneth S. Davis, M.D. (Los Angeles): The series of papers which we have just heard provides a stimulating example of the interest which the groups from the Mayo Clinic and Philadelphia have had in the roentgen manifestations of diseases of the skull and intracranial contents. We who are practising general radiology have become accustomed to relying upon papers such as these for guidance and instruction.

It is proper to re-emphasize, at this time, the fact that in order to accomplish correct roentgen interpretation we must have the most perfect roentgen technic that our diligence and equipment will permit; otherwise many of the roentgen conditions described today will be overlooked. Roentgenograms of the skull of good technical quality are not always easy to obtain, and we must not be satisfied until we have perfected ourselves in such technics as have been described by the essayists.

The importance of the changes in and around the sella turcica have been well emphasized today by the frequent allusions made to them. The nature of the changes, both direct and indirect, have been well described. There would appear, however, to be some uncertainty, at least to me, in the correlation of the actual sellar changes with the terminology used to describe them. I wonder if Dr. Camp would try to clarify for us the use of the terms atrophy, demineralization, erosion, and destruction. Certainly destructive changes are often present. On the other hand, Dr. Camp has shown us that changes which are called destructive are in reality not so, since recalcification can occur.

Dr. Pendergrass has pointed out the difficulty in the early recognition of meningiomas, particularly those around the sphenoidal ridge. This is important in view of the fact that, while meningiomas can be cured if completely removed, they are so frequently discovered in such an advanced state that complete removal is difficult, if not impossible. It behooves us, therefore, to bend every effort to recognize these lesions as promptly as we can.

In so far as the differentiation of changes suggestive of meningiomas is concerned, the helpfulness of arteriography should not be forgotten since, as Lorenz and others have pointed out, the angio-arteriographic findings may be of great value.

Probably most of us have had the impression that the roentgen examination in patients with tumors of the 8th nerve has been disappointing. Dr. Hodes' indication, therefore, that abnormal findings are present in around 80 per cent of patients is a definite stimulus for us to perfect our technic and diagnostic acumen so that we, too, can recognize early changes produced by these and other tumors in this region.

Fray has devised a proportional method of localizing the pineal gland which incorporates the use of an elastic cord upon which are placed markers which define the normal zones of the visible gland in both the long and vertical diameters of the skull, which is of valuable aid to the general

radiologist. I would appreciate it if Dr. Young would elaborate on this method in his closing discussion.

Curtis H. Burge, M.D. (Houston, Texas): I would like to make a few points in relation to Dr. Young's paper. First of all, since the pineal is more or less in the center of the head, we are able to recognize lateral displacements in films which are rotated. Of course we want perfectly straight projections, but we don't always get them, especially in patients who are disoriented and uncooperative. Considerable rotation of the head can be present and not produce a false appearance of displacement of the pineal to one side. When there is a borderline case, it is always necessary to verify the impression of lateral displacement on more than one projection. Therefore, we like to see it on the occipital projection as well as on the postero-anterior.

I believe that Dr. Young talked about displacement of the pineal in connection with space-occupying lesions. It should also be remembered that atrophic lesions of the brain which are quite severe can cause the pineal to shift toward the side of the lesion, so that it is not always indicative of a space-occupying disease.

Routine measurements of the distance between the pineal and the side walls of the skull should always be made. In every film that we interpret where we can see the calcified pineal, we should actually measure those distances with a ruler; if we depend simply upon estimation of the midline position of the pineal, we may make some pretty serious mistakes. We should always remember that there is a certain amount of normal variation between the measurements on the two sides, but I believe that Dr. Young's figure, over 2.5 mm. displacement to one side, is practically always indicative of abnormality.

Displacement of the falx is, I think, absolutely unreliable and should not be depended upon, because, as has been pointed out in this discussion, the falx is a rigid structure which is not easily displaced. In ventriculograms of large lesions in the cerebral hemispheres, the falx will remain more or less in the midline and the lateral ventricle on the side of the lesion will be displaced under it. That was illustrated on Dr. Pendergrass' ventriculogram of a sphenoid ridge meningioma with calcification of the falx.

Merrill C. Sosman, M.D. (Boston, Mass.): I would like to put in just a sentence on two or three matters. I certainly would agree that displacement of the falx is very, very rare indeed, if it occurs at all. Personally, I have not seen displacement of the falx by space-occupying tumors. The brain slides under the falx, but the falx remains rigid.

Secondly, the meningiomas are the most outstanding example of tumor arising following an incident trauma. A large number of Dr. Cushing's patients had very definite scars in the scalp directly over the location of the tumor, and those scars were often not recognized, not known, and not identified until the scalp had been shaved preliminary to operation.

The third thing is that I recommend that all of you, if you have not already done so, purchase that monograph on meningiomas by Cushing and Eisenhardt. To me, it is the best existing example of the perfect monograph. Dr. Cushing spent two years of his life writing it after he had retired from Harvard. The monograph is complete in every detail except one, and that is the description of all of the variations of the radiological pictures that we see. Dr. Cushing may have left that for me.

If Dr. Pendergrass is still here, I would like to pick on him a little bit from the point of view of the Boston purist. I heard him use the word "hypophysis" when he meant "sella." Now, to me hypophysis is synonymous with the pituitary body. If he had used, "hypophyseal fossa," I think that would have been correct.

The final point that I would like to make is that we do not trust rotated anteroposterior or postero-anterior films for displacement of the pineal. The skulls of patients, as I can see by looking over the audience, vary tremendously. Some are round, some are oval, some are egg-shaped, and an unusually protruding frontal bone may throw the measurements off considerably if there is just a small degree of rotation. I insist, before making a diagnosis of a lateral displacement of the pineal, that we have films as nearly perfect as possible. In many cases, a moderate degree of rotation does not make a difference, but you never know which one will be critical. Along that same line, remember that some skulls are asymmetrical from birth, possibly from birth injuries or from a slight difference in the development of the two sides during the growing period of the individual.

Dr. Young (*closing*): I agree with Dr. Sosman that rotation of the head must be avoided. Exact positioning and good quality roentgenograms are essential in the study of pineal position.

It is true that the pineal is not in the midline when there is asymmetry on the basis of unilateral underdevelopment of the skull. It is found near the lateral wall of the small half of the cranium because the cerebral hemisphere on this side is smaller than the normally developed hemisphere. The unusual position of the pineal must not be erroneously attributed to space-taking disease.

The falx is rarely a valuable landmark in the determination of space-occupying disease unless extensively calcified when displacement is demonstrable [See Fig. 7, p. 630].

Spinal Cord Tumors[1]

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SINCE 60 TO 85 PER CENT (1, 2) of spinal cord tumors are benign and potentially curable, it is of the utmost importance to establish the diagnosis before irreversible pressure changes have occurred in the cord. Our review of 68 verified cases indicates, however, that early diagnosis is infrequent. We believe that the reason for the common delay in making an early diagnosis of spinal cord tumor is failure on the part of many physicians to consider this possibility.

Tumors of the spinal cord are not common. At the University of Chicago Clinics, only 58 cord tumors were diagnosed in 234,000 admissions over a ten-year period (3). In a similar period at the Cleveland Clinic, one verified cord tumor has been recorded for approximately every 2,518 patients examined. It is believed that early diagnosis of spinal cord tumors is possible in at least 95 per cent of the cases if careful clinical and roentgenologic investigations are conducted. In our series, 48 per cent of the patients with this diagnosis had spastic or flaccid paralysis and sphincter disturbances when first seen at the Clinic; this suggests that the diagnosis could have been made much sooner.

MATERIAL

This study is concerned only with primary tumors arising within the spinal canal and beneath the dura. Extradural tumors have been eliminated from consideration unless exploration established the fact that they originated beneath the dura and subsequently split it to occupy an extradural position.

The histologic classification of the 71 tumors removed from 68 patients is presented in Table I. As is usual the neurofibromas outnumbered all other cell types.

TABLE I

Neurofibroma	30
Meningioma (occurring in 19 patients)	22
Ependymoma	10
Astrocytoma	3
Glioblastoma multiforme	2
Glioma (unclassified)	1
Mixed tumor	1
Epidermoid	1
Hemangioblastoma	1
TOTAL	71

Meningiomas and ependymomas followed in order of frequency.

Foerster and Bailey (4) reported two instances of multiple spinal cord tumors in a series of 100 cases, and Camp (5) has indicated that 4 per cent of spinal cord tumors are multiple. In our group there was a patient who had one subdural meningioma removed from the region of D-8 in July 1933, a second meningioma removed at D-10 in November 1933, a third removed from D-6 in July 1946, and a fourth from D-9 in September 1946.

The series is somewhat unusual in that it contained 2 developmental tumors—an epidermoid and a mixed tumor. To date approximately 80 developmental tumors have been reported in the literature.

Of the 71 tumors, 48 were subdural; 11 were subdural and extradural; 12 were intramedullary. Our incidence of intramedullary tumors—16 per cent—is slightly higher than the 11.5 per cent reported by Rasmussen, Kernohan, and Adson (6), but it is within the limits of 10 to 20 per cent quoted by others (7).

The distribution of tumors within the spinal canal was not unusual. Sixteen were in the cervical spine, 29 in the dorsal, 25 in the lumbar, and 1 in the sacral spine. As in most reported series of cord tumors, meningiomas predominated in the dorsal spine. Seventeen of our 22 meningiomas were dorsal.

[1] From the Cleveland Clinic and the Frank E. Bunts Educational Institute. Presented at the Thirty-fourth Annual Meeting of the Radiological Society of North America, San Francisco, Calif., Dec. 5–10, 1948.

CLINICAL ASPECTS

Age: Our patients ranged in age from 9 to 82 years. The mean average age was 40. Sixty-four per cent of the patients were evenly distributed in the third, fourth, and fifth decades.

Sex: Forty-one of the 68 patients were women. This predominance of women is of questionable significance, since in other reported series the sex distribution is nearly equal. It is significant that 17 of the 22 meningiomas were removed from women, and also that all of the meningiomas in women were located in the dorsal spine.

Duration of Symptoms: The mean average time from onset of symptoms to an established diagnosis of cord tumor was twenty-four months. In the more rapidly growing glioma group, the diagnosis was occasionally established two or three months after the onset of symptoms.

Symptoms: If there is one symptom that might be called characteristic of cord tumors, it is pain. In 50 of our patients the first symptom was pain and 5 others complained of pain later in the course of the disease. Twelve of the 50 had neurologic disturbances coinciding with the onset of pain. The pain was usually described as burning or lancinating. It usually began in the neck, between the shoulders, or in the lower back, but it frequently radiated down one or both arms or one or both legs. Generally, when the pain was unilateral at the start, it became bilateral before long. In the case of those tumors situated in the cervical cord, there were frequently symptoms and neurologic signs relating to all four extremities at the time of operation.

Occasionally the pain radiated around the thorax or abdomen in a girdle-like distribution. In most of our patients it was exaggerated by coughing or sneezing; as a rule, it was decreased on standing or walking. Nearly 25 per cent of the patients volunteered the information that they had been sleeping in a chair for months because sleeping in bed intensified the pain.

Thirty patients had neurologic symptoms from the onset. The most common of these were paresthesias, usually described as numbness and tingling or sensations of heat and cold. An additional 25 patients had neurologic changes later in the disease. In this group, stiffness or weakness of the back or arms or legs, often associated with muscle atrophy, was most common. Depending on the level of the tumor and its situation in the spinal canal, the deep reflexes of the upper and lower extremities were either exaggerated, hypoactive, or absent. There were frequent disturbances of vibratory and position sense, and the sensations of pain, heat, and cold were nearly always altered in those patients who were paralyzed on admission to the Clinic.

Intramedullary tumors could not be differentiated from extramedullary ones on the basis of symptomatology. Eight of the 12 patients having intramedullary tumors had both pain and neurologic disturbances before operation. Three of the 8 had pain as the first symptom, and 5 dated their neurologic disturbances from the very start. There were only 4 patients who had no pain during the course of the disease up to the date of operation.

Past History: Many of the patients had had repeated courses of physical therapy before the correct diagnosis was established. Listed among the previous treatments were several lumbosacral fusions, an abdominal exploration for persistent unexplained pain, a nephropexy, a rib and intercostal nerve resection, and an application of a body cast for arthritis. This supports the contention of Adson (8) that 10 per cent of patients eventually diagnosed as having cord tumor have had operations for some thoracic or abdominal lesion without relief of pain.

LABORATORY DATA

The importance of a spinal fluid examination in the diagnosis of cord tumor cannot be overemphasized. Lumbar puncture should be done at the interspace between the fifth lumbar vertebra and the sacrum, since most tumors will lie cephalad to that point. By means of jugular com-

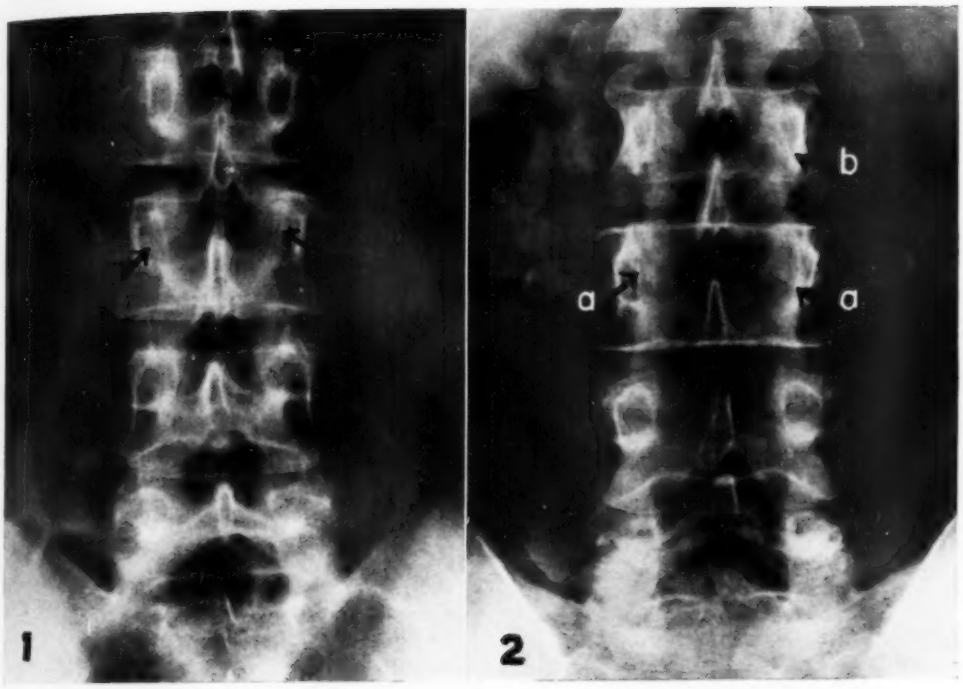

Fig. 1. Neurofibroma. Beginning erosion, medial aspect of both pedicles, L-3.
Fig. 2. Mixed tumor. a. Erosion, medial aspect of both pedicles, L-3. b. Early erosion, medial aspect of left pedicle, L-2.

pression and manometric studies of spinal fluid pressure, it is nearly always possible to establish the presence of partial or complete subarachnoid block. The presence or absence of arterial pulsations and respiratory movements in the spinal fluid are likewise important observations during spinal fluid examination. About 25 per cent of our patients had combined lumbar and cisternal punctures for a more complete study of spinal fluid dynamics.

Spinal fluid protein studies are extremely important. Total protein values above 40 mg. per cent are indicative of a partial or complete subarachnoid block in the absence of degenerative or inflammatory disease of the cord or brain. Forty-eight of our 59 patients on whom this test was performed had definite elevation of spinal fluid protein. In 34 instances the protein was over 100 mg. per cent and 12 values were over 1,000 mg. per cent. Elevated spinal fluid protein is not pathognomonic of spinal cord tumor, but in the presence of sensory changes and partial or complete spinal subarachnoid block the evidence is overwhelmingly in favor of this diagnosis.

ROENTGENOLOGIC CONSIDERATIONS

Plain Films: Camp (9) estimated that 21.6 per cent of intradural extramedullary spinal cord tumors caused erosion or infiltration of the spinal canal. In our series, roentgenograms were available for study on all patients. We were able to detect diagnostic evidence on the plain films in only 11 of the 68. This represented 16 per cent of the series. All of the bone changes, however, were in the intradural extramedullary group, so that 19.6 per cent of that group had positive roentgen evidence on plain films. The only diagnostic changes observed by us were erosions or infiltration of the bony canal.

Although Gray (10) has reported that somewhat less than 10 per cent of spinal

Fig. 3. Epidermoid. A. Anteroposterior projection. a. Extensive erosion of pedicles and laminae, L-3. b. Beginning erosion of pedicles, L-2 and L-4. B. Lateral film. Erosions of posterior surface, L-2 and L-3.

Fig. 4. Neurofibroma. A. Erosion of vertebral bodies and lateral masses of C-3 and C-4. B. Defect demonstrated on lateral film.

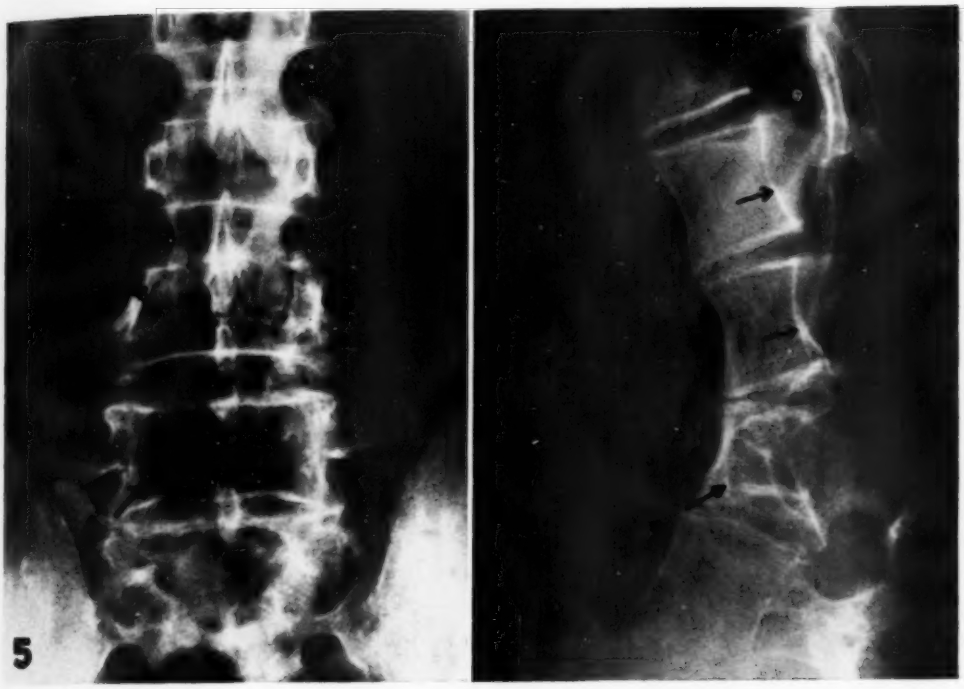

Fig. 5. Invasive meningioma. Infiltrative destruction of vertebrae, laminae, and pedicles, L-4 and L-5.
Fig. 6. Neurofibroma. Extensive erosion of vertebrae, pedicles, and laminae, L-1, L-2, and L-3.

cord tumors show calcification, we were unable to detect any calcification in our tumors roentgenologically, even though several showed gross calcification at exploration. Calcification is apparently uncommon except in meningiomas, occasional hemangioblastomas, and vascular oligodendrogliomas.

We agree with Camp (9) that actual measurement of the interpedicular spaces, according to the technic of Elsberg and Dyke (11), is not as important as careful inspection of the pedicles and bony canal. In all of our cases showing bone erosion, the defects were evident to the roentgenologist without recourse to charts of the interpedicular distances. Subsequently films on all 68 patients were reviewed, and interpedicular spaces were measured and charted. We were unable to detect any additional cases of bone erosion.

The earliest bone change was erosion of the medial aspect of the pedicles (Fig. 1). A number showed rather symmetrical erosion of the medial aspects of both pedicles in several vertebrae (Fig. 2). As the tumors enlarge, they eventually produce pressure erosion of the spinal canal, with atrophy of the pedicles, laminae, and posterior surfaces of the vertebrae (Fig. 3). In the cervical spine, punched-out defects (Fig. 4) in the lateral masses and enlargement of the intervertebral foramina are not uncommon.

Subdural and intramedullary tumors do not, as a rule, invade bone; however, we have seen an invasive meningioma that extensively infiltrated several vertebral bodies (Fig. 5) and their laminae, pedicles, and spinous processes.

Dyke (12) has pointed out that multiple vertebral erosions are unusual except in ependymomas, congenital cysts, and giant tumors of the cauda equina. One of our neurofibromas (Fig. 6) caused rather extensive changes in the first three lumbar vertebrae. Furthermore, both of our developmental tumors eroded several vertebrae.

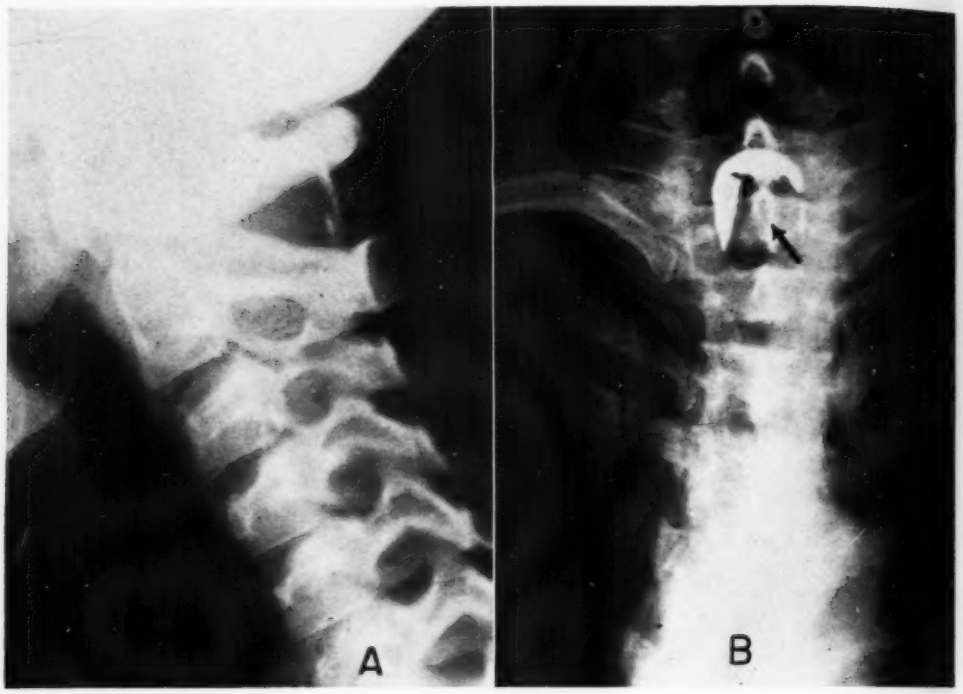

Fig. 7. A. Intramedullary astrocytoma, C-1 and C-2. Cisternal myelogram revealing increased diameter of the cord due to tumor. B. Intramedullary ependymoma. Cisternal myelogram. Partial obstruction with central defect, increased width of cord and lateral extension of oil along tumor.

Extraspinal extensions of cord tumors occasionally are demonstrated roentgenologically. The extradural extension of the dumbbell-type neurofibroma is the best example. We have found these soft-tissue extensions difficult to identify in the cervical spine but rather easy to detect along the dorsal spine. In the lumbar area they can be seen occasionally indenting the psoas muscle shadow.

MYELOGRAPHY

Camp (5) and others have pointed out that opaque media should not be used in the spinal canal until all other methods of diagnosis of spinal cord tumors have been attempted. Horrax (13) contends that a myelogram should be done only when the level of a lesion cannot be determined from neurologic studies. Gardner (14) believes that any patient suspected of having a cord tumor is entitled to a myelogram to establish or exclude the diagnosis, unless either the plain roentgenogram or the physical examination is unequivocal. Delay in the diagnosis of spinal cord tumors is in part attributable to the reluctance of many physicians to order myelograms on their patients.

Our best myelograms have been obtained with thorotrast. This medium was used for all lumbar myelograms done at the Cleveland Clinic for a period of three of four years, until several severe meningeal reactions were observed for the first time in 1943. Since then, pantopaque has been used routinely. We have had practically no experience with air myelography.

We believe that lumbar myelography is the procedure of choice when there is no evidence of subarachnoid block. We prefer to use 5 c.c. of opaque medium. Our technic combines roentgenoscopy, spot roentgenograms, and survey films as necessary to demonstrate each lesion to best advantage.

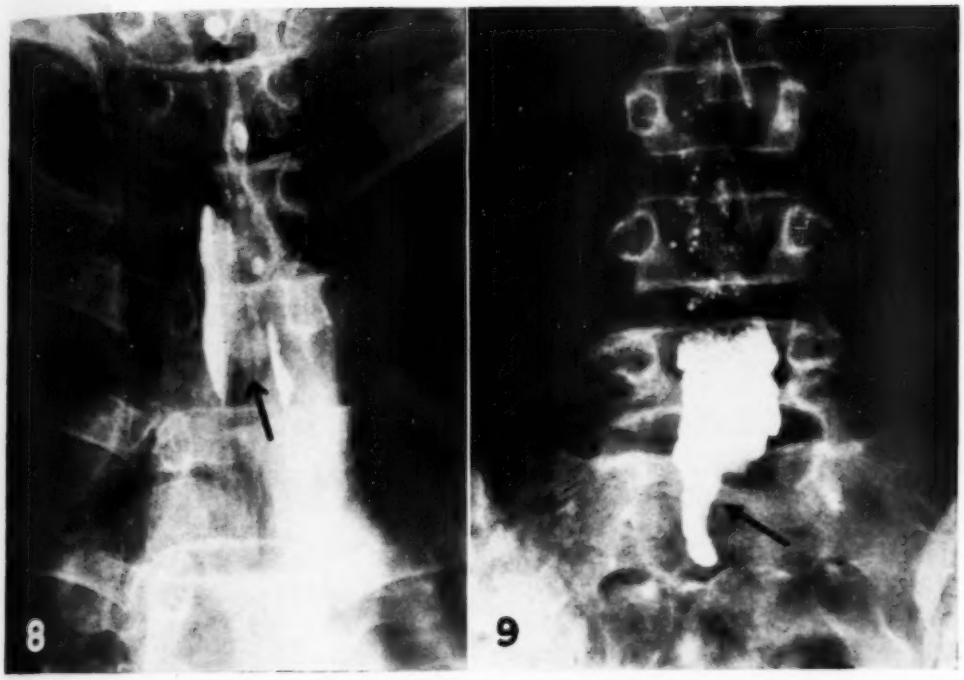

Fig. 8. Extradural Hodgkin's paragranuloma. Cisternal myelogram. Partial obstruction of lower border, D-5, with central defect and lateral extension of oil.
Fig. 9. Intradural and extradural neurofibroma. Lumbar myelogram. Lateral defect, S-1.

When a subarachnoid block has been demonstrated by a study of spinal fluid dynamics, our neurosurgeons inject oil into the cisterna magna. They reason that, since a block has been demonstrated, the prime purpose of the myelogram is to identify the upper level of the lesion. From the standpoint of excision they are interested in the upper limit of the tumor. The lower limit is often determined with reasonable accuracy by the neurologic studies, whereas the upper margin is seldom sharply defined. Furthermore, since surgical exploration is almost always indicated in the presence of subarachnoid block, the identity of the lesion is of secondary importance. This explains the large number of cisternal myelograms, 29, as compared to lumbar myelograms, 11, in our series.

We have used as little as one or two drops of oil in cisternal myelography and have seldom used more than 3 c.c. Despite this small amount, our tumor localization has been correct in 27 of 29 cisternal myelograms. One failure occurred on the fifth myelogram in a patient who had multiple meningiomas. In this case, three previous dorsal laminectomies, old oil in the subarachnoid space, and arachnoidal adhesions combined to make the task more difficult. There were no errors of localization in the lumbar myelograms. Our accuracy, 94.6 per cent, compares favorably with that of Camp (5), 96.8 per cent.

The character of the defects produced by intramedullary tumors have been described by others (5, 15, 16). They have pointed out that partial block, widening of the cord, and lateral displacement of the oil are rather characteristic of intramedullary tumors (Fig. 7). We have occasionally seen a somewhat similar appearance with extradural lesions (Fig. 8).

Lateral defects (Fig. 9) in the opaque oil are more often seen in those tumors which have arisen beneath the dura and have

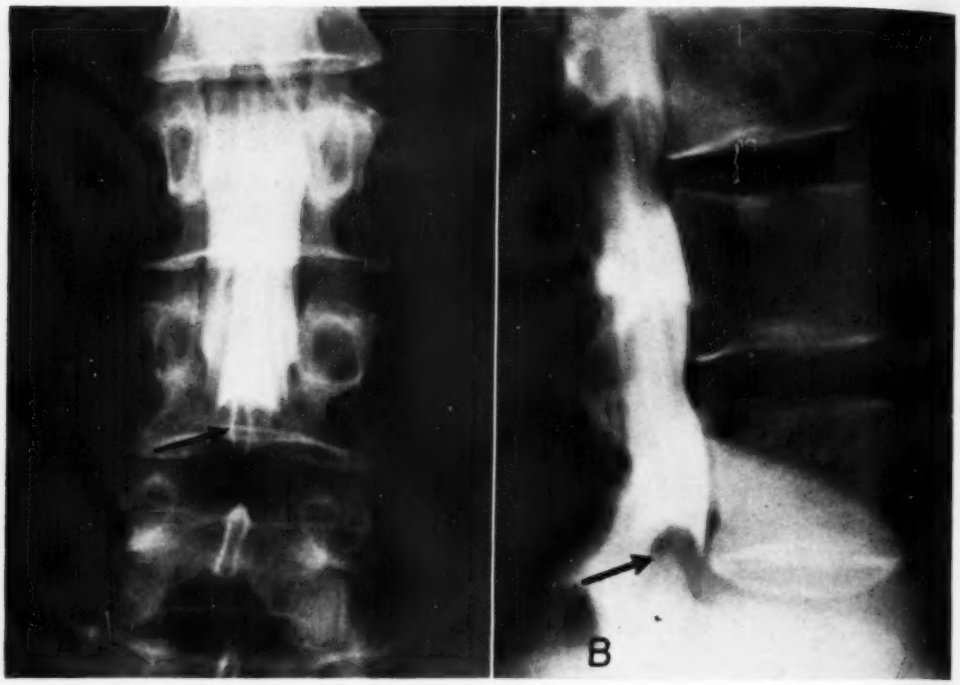

Fig. 10. Neurofibroma. Thorotrast myelogram. A. Anteroposterior view. B. Lateral view. Complete obstruction at lower border, L-4, with central concave defect.

grown through the dura and into an intervertebral foramen.

When a subdural tumor produces a complete block, there is a central concave defect (Fig. 10) in the opaque oil column. This was characteristic of a good number of our subdural tumors.

PROGNOSIS

The elapsed time following operation has been too short in many of our patients to permit definite conclusions about prognosis. As would be expected, however, patients with the more malignant types of intramedullary gliomas have done poorly. Two died in two to four months after operation. One patient having an hemangioblastoma died twenty-seven months after operation. On the other hand, patients with intramedullary ependymomas have fared better. One is alive without recurrence after five years. Most of the patients with neurofibromas and meningiomas have survived, but the degree of recovery has been directly related to the extent of cord damage and the duration of symptoms before removal of the tumor. Hyndman and Russell (17) have emphasized that it is not always possible to predict the degree of recovery prior to surgery, but the results are often surprisingly good.

SUMMARY

1. Delay in the diagnosis of cord tumors is due primarily to a low index of suspicion.

2. Myelography is indicated in any cord tumor suspect on whom the diagnosis and localization cannot be established by other means.

3. In our series, myelography localized the tumor accurately in 94.6 per cent of the cases in which it was attempted.

4. Degree of recovery in spinal cord meningiomas and neurofibromas is directly related to the degree of irreversible cord damage.

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REFERENCES

1. ADSON, A. W.: Intraspinal Tumors; Surgical Consideration; Collective Review. Internat. Abstr. Surg. **67:** 225–237, 1938, in Surg., Gynec. & Obst., September 1938.
2. BUNTS, A. T.: Tumors of Spinal Cord. Proc. Interstate Postgrad. M. A. North America (1943), pp. 156–158, 1944.
3. WALKER, A. E.: Early Diagnosis of Spinal Cord Tumor. J. Indiana M. A. **33:** 360–366, July 1940.
4. FOERSTER, O., AND BAILEY, P.: Cited by Lichtenstein, B. W.: Multiple Primary Tumors of Spinal Cord. Arch. Neurol. & Psychiat. **46:** 59–71, July 1941.
5. CAMP, J. D.: Contrast Myelography. M. Clin. North America **25:** 1067–1102, July 1941.
6. RASMUSSEN, T. B., KERNOHAN, J. W., AND ADSON, A. W.: Pathologic Classification, with Surgical Consideration, of Intraspinal Tumors. Ann. Surg. **111:** 513–530, April 1940.
7. SHENKIN, H. A., AND ALPERS, B. J.: Clinical and Pathologic Features of Gliomas of Spinal Cord. Arch. Neurol. & Psychiat. **52:** 87–105, August 1944.
8. ADSON, A. W.: Tumors of Spinal Cord: Diagnosis and Treatment. Canad. M. A. J. **40:** 448–454, May 1939.
9. CAMP, J. D.: Roentgenologic Localization of Tumors Affecting Spinal Cord. Am. J. Roentgenol. **40:** 540–544, October 1938.
10. GRAY, E. D.: Calcification and Ossification of Spinal Tumours. Brit. J. Radiol. **15:** 365–369, December 1942.
11. ELSBERG, C. A., AND DYKE, C. G.: Diagnosis and Localization of Tumors of Spinal Cord by Means of Measurements Made on X-ray Films of Vertebrae, and Correlation of Clinical and X-ray Findings. Bull. Neurol. Inst. New York. **3:** 359–394, March 1934.
12. DYKE, C. G.: Roentgen-ray Diagnosis of Spinal Cord Tumors. In Diagnostic Roentgenology, edited by R. Golden, New York, Thomas Nelson and Sons, Vol. II, pp. 561–582.
13. HORRAX, G.: Extramedullary Spinal Cord Tumors. S. Clin. North America **27:** 535–553, June 1947.
14. GARDNER, W. J.: Personal communication.
15. EPSTEIN, B. S., AND DAVIDOFF, L. M.: Myelographic Diagnosis of Extramedullary Cervical Spinal Cord Tumors. Am. J. Roentgenol. **55:** 413–419, April 1946.
16. WALKER, A. E., JESSICO, C. M., AND MARKOVICH, A. W.: Myelographic Diagnosis of Intramedullary Spinal Cord Tumors. Am. J. Roentgenol. **45:** 321–331, March 1941.
17. HYNDMAN, O. R., AND RUSSELL, E. P.: Tumors of Spinal Cord; Some Clinical Considerations: Recoverability of Cord Function. J. Iowa M. Soc. **28:** 425–435, September 1938.

SUMARIO

Tumores de la Médula Espinal

Este repaso versa sobre una serie de 71 tumores de la médula espinal extirpados a 68 enfermos, comprendiendo: 30 neurofibromas, 22 meningiomas, 10 ependimomas, 3 astrocitomas, 2 casos de glioblastoma multiforme y 1, cada uno, de tumor mixto, epidermoide y hemangioblastoma. En los tumores de la médula espinal el síntoma sobresaliente es el dolor, habiendo sido también el inicial en 50 enfermos de esta serie y aparecido más tarde en otros 5. En 55 pacientes hubo al principio o más tarde síntomas neurológicos. Muchos de los enfermos habían sido tratados sin resultado con la fisioterapia u otras medidas antes de establecerse el diagnóstico.

Roentgenológicamente, la patología ósea más temprana consistió en erosión de la cara medial de los pedículos. Al agrandarse los tumores, produjeron con el tiempo erosión hipertensiva del conducto raquídeo, con atrofia de los pedículos, láminas y caras posteriores de las vértebras. En la porción cervical del raquis, no son raras las deformaciones excavadas de las masas laterales y la hipertrofia de los agujeros intervertebrales.

A los AA. la mielografía lumbar les ha resultado ser el procedimiento de elección para el descubrimiento de estos tumores cuando no hay signos de bloqueo subaracnoideo. Si el estudio de la dinámica del líquido raquídeo indica bloqueo subaracnoideo, está indicada la mielografía cisternal. Aunque los mejores mielogramas son los obtenidos con torotrasto, se ha abandonado el empleo de este medio debido a la aparición de algunas graves reacciones meníngeas. El pantopaco es empleado ahora sistemáticamente. En 94.6 por ciento de los casos de esta serie en que se probó la mielografía, localizó el tumor con exactitud. Está indicado este procedimiento en todo enfermo en que se sospecha tumor medular y no pueden establecerse por otros medios el diagnóstico y la localización.

En los meningiomas de la médula espinal el grado de reposición guarda relación directa con el grado de las lesiones medulares irreversibles.

Prepyloric Local Gastritis[1]

JAMES B. HAWORTH, M.D., and NOEL B. RAWLS, M.D.

SPASM AND MUCOSAL deformity in the prepyloric segment of the stomach are among the most common abnormal manifestations observed by radiologists in examination of the upper gastro-intestinal tract. Antral gastritis has been described in detail by Golden (4), and the radiographic findings have been elaborated by Arendt (9). Forssell (1) performed exhaustive studies of the radiographic appearance of the mucous membrane of the gastro-intestinal tract. Wolf and Wolff (5) reported observations on healthy gastric mucosa, made in a patient with a large gastrostomy. It will be our purpose here to correlate the findings of these investigators with those in our own series of 1,519 upper gastro-intestinal studies performed on 1,289 patients, with special reference to an abnormal state of the distal segment of the stomach which we call "prepyloric local gastritis."

TABLE I: INCIDENCE OF PREPYLORIC LOCAL GASTRITIS IN 1,289 PATIENTS

Prepyloric local gastritis, uncomplicated (*i.e.*, without demonstrable ulcer)	109 cases (8.5%)
Prepyloric local gastritis, associated with other gastro-duodenal abnormalities	176 cases (13.7%)
No evidence of prepyloric local gastritis	1,004 cases (77.8%)
TOTAL	1,289 cases (100%)

This redundant term was selected to emphasize the primarily local nature of the disturbance, as contrasted with the more generalized types of gastritis which have been described frequently by gastroscopists. Our attention was directed to this condition by a case exhibiting a fixed prepyloric deformity thought to be of neoplastic origin, in which no organic disease was found at laparotomy. Less marked degrees of antral irregularity were seen in many patients exhibiting no demonstrable organic lesion. Through clinical study of these cases, a characteristic syndrome emerged. Pathologic material for histologic examination has been scanty because of the rarity of surgical intervention in these cases, but sufficient material has been obtained in 9 cases to indicate that abnormal changes occur in the gastric mucosa in this disease.

INCIDENCE

The incidence of prepyloric local gastritis in our series is shown in Table I. A more detailed analysis of the relative frequency of this condition, occurring alone and in combination with other abnormalities of the upper gastro-intestinal tract, is given in Table II. In this latter table the sex incidence is also shown.

It is apparent that prepyloric local gastritis is slightly less frequent in occurrence than duodenal ulcer, but is more than twice as frequent as gastric ulcer. There seems to be no significant difference in incidence between the sexes. Cases have been observed as early as seven years of age and as late as seventy-eight years. The peak incidence is in the third decade.

ETIOLOGY

It is our opinion that prepyloric local gastritis is primarily a psychosomatic disorder in which the parasympathetic (craniosacral autonomic) nervous system is subjected to excessive stimulation of central origin. The vagus nerves mediate both motor activity and secretion in the stomach. The beneficial effect of vagotomy in some cases of intractable peptic ulcer appears to be established. The administration of atropine, which blocks the nerve endings of the parasympathetic (craniosacral autonomic) system, diminishes or relieves the symptoms of these patients. Smoking, on the other hand, aggravates the complaints and appears to

[1] Accepted for publication in November 1948.

TABLE II: SEX INCIDENCE OF PREPYLORIC GASTRITIS

	Males	Females	All Cases
Entire series	714 (100%)	575 (100%)	1,289 (100%)
Prepyloric local gastritis			
Alone	55 (7.7%)	54 (9.4%)	109 (8.5%)
With duodenal ulcer	54 (7.6%)	24 (4.2%)	78 (6.0%)
With gastric ulcer	25 (3.5%)	17 (3.0%)	42 (3.3%)
With duodenal and gastric ulcer	21 (2.9%)	6 (1.0%)	27 (2.1%)
With duodenal deformity (due to healed duodenal ulcer)	5 (0.7%)	13 (2.3%)	18 (1.4%)
With duodenitis	7 (1.0%)	4 (0.7%)	11 (0.9%)
Total cases of prepyloric local gastritis	167 (23.4%)	118 (20.5%)	285 (22.1%)
Duodenal ulcer (all cases)	216 (30.3%)	102 (17.5%)	318 (24.7%)
Gastric ulcer (all cases)	72 (10.1%)	56 (9.7%)	128 (9.9%)
Normal patients (including non-inflammatory abnormalities)	316 (44.3%)	353 (61.5%)	669 (51.9%)

delay recovery: nicotine paralyzes the sympathetic ganglia in Auerbach's plexus, and may have an effect similar to that of stimulation of the antagonists, on the parasympathetic (craniosacral autonomic) nervous system.

Patients exhibiting the characteristic clinical syndrome and radiographic findings of prepyloric local gastritis are quite commonly of the "nervous" type. Histories often reveal recent traumatic episodes or situations involving abnormal stress upon the emotions or capabilities. We have been able to follow a number of cases for periods up to two years, with serial radiographic studies at intervals of three months or less. In these patients, periods of emotional stress were usually characterized by exacerbations, both symptomatically and objectively, while in periods of tranquillity remissions occurred. In this connection, the following quotations are of interest (Portis):

"A normal digestive tract cannot have continuous emotional stimuli reach it day after day, week after week, month after month, and even year after year, and still remain normal without showing evidence of physiologic and structural change. We, however, have failed to recognize these changes. Increasing clinical experience, with laboratory and roentgenologic evidence, must show some deviation from normal if the patient has symptoms."

"Any gastroenterologist with a broad clinical experience will honestly admit that a majority of his patients have a symptom complex for which no organic basis can be found."

We believe that many of these patients have prepyloric local gastritis.

The observations of Wolf and Wolff (5) on the exposed intact gastric mucosa in the gastrostomy of a patient with esophageal obstruction, contribute to an understanding of the mechanism present. The following sentences are especially pertinent:

"The hypersecreting stomach is always hyperemic, turgid, engorged."

"This is true, regardless of whether the accelerated secretion occurred in response to the stimulus of food, alcohol, or histamine, or to certain emotionally changed situations."

"When the hyperfunctioning, engorged state was prolonged in our subject, he frequently complained of abdominal discomfort and pain."

SYMPTOMATOLOGY

The most characteristic complaint in this group of patients is distress, which may be of a burning, aching, or griping character, generally occurring from fifteen to thirty minutes after the ingestion of food. The discomfort is localized to the epigastrium or lower sternum; it is frequently relieved by alkalies or demulcent agents. Spices, alcohol, and coffee aggravate the condition, while a bland diet relieves the symptoms, entirely or in part. Some of the cases exhibit the typical ulcer sequence of distress-food-relief, or there may be discomfort both after eating and when the stomach is empty. Gastrointestinal hemorrhage has occurred in uncomplicated cases of prepyloric local gastritis. In 13 patients, 1 per cent of our entire series of cases, there was gastrointestinal bleeding for which no other organic cause could be found. This represents 12 per cent of our cases of uncomplicated prepyloric local gastritis.

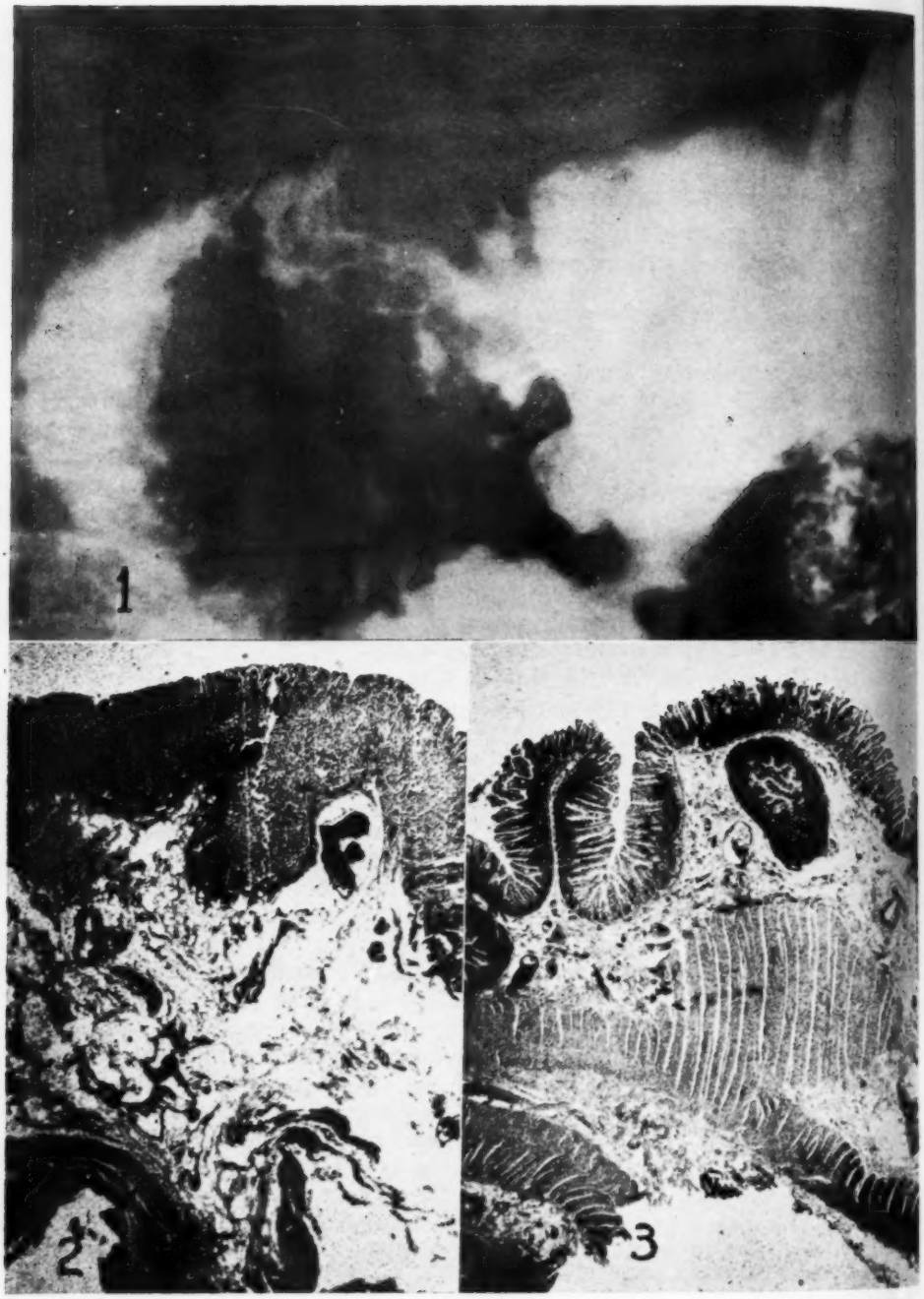

Plate I: Prepyloric local gastritis: Preoperative roentgenogram (1) and low-power photomicrographs of the gastric wall (2 and 3).

2. Distal segment of the stomach. Note the increased thickness of the gastric mucosa. Higher magnification revealed lymphocytic infiltration of the mucosal layer only. Note also the edema of the submucosal layer.
3. Pars media, same magnification as 2. Note the normal mucosal layer and the compact submucosa.

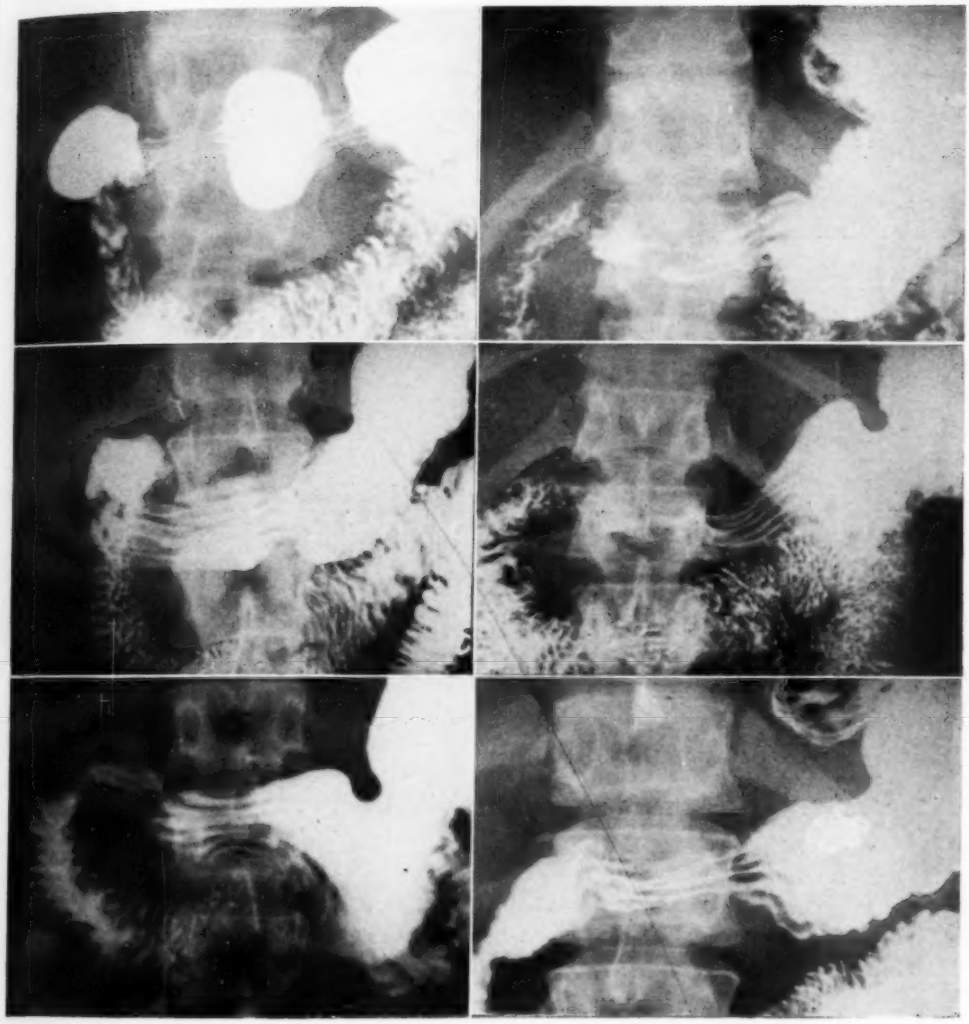

Plate II: Normal mucosal folds in the prepyloric segment of the stomach.

PATHOLOGY

The available material indicates that there is a pronounced infiltration of the mucous membrane with lymphocytes. This process, in our series, is limited to the mucosa, and there is no invasion of the submucosa or muscularis. Edema of the submucosal connective tissue is, however, a frequent finding. The mucosa is increased in thickness: there is diapedesis or capillary hemorrhage in the mucous membrane. Ulceration is commonly absent, but there may be superficial erosion of the gastric mucosa. The photomicrographs in Plate I illustrate the findings in the prepyloric area, with normal gastric tissue from the pars media for comparison. The preoperative roentgenogram of the patient's stomach is included.

ROENTGEN DIAGNOSIS

The belief seems widely established that the roentgenologic diagnosis of gastritis is not reliable. Gastroscopists generally scoff

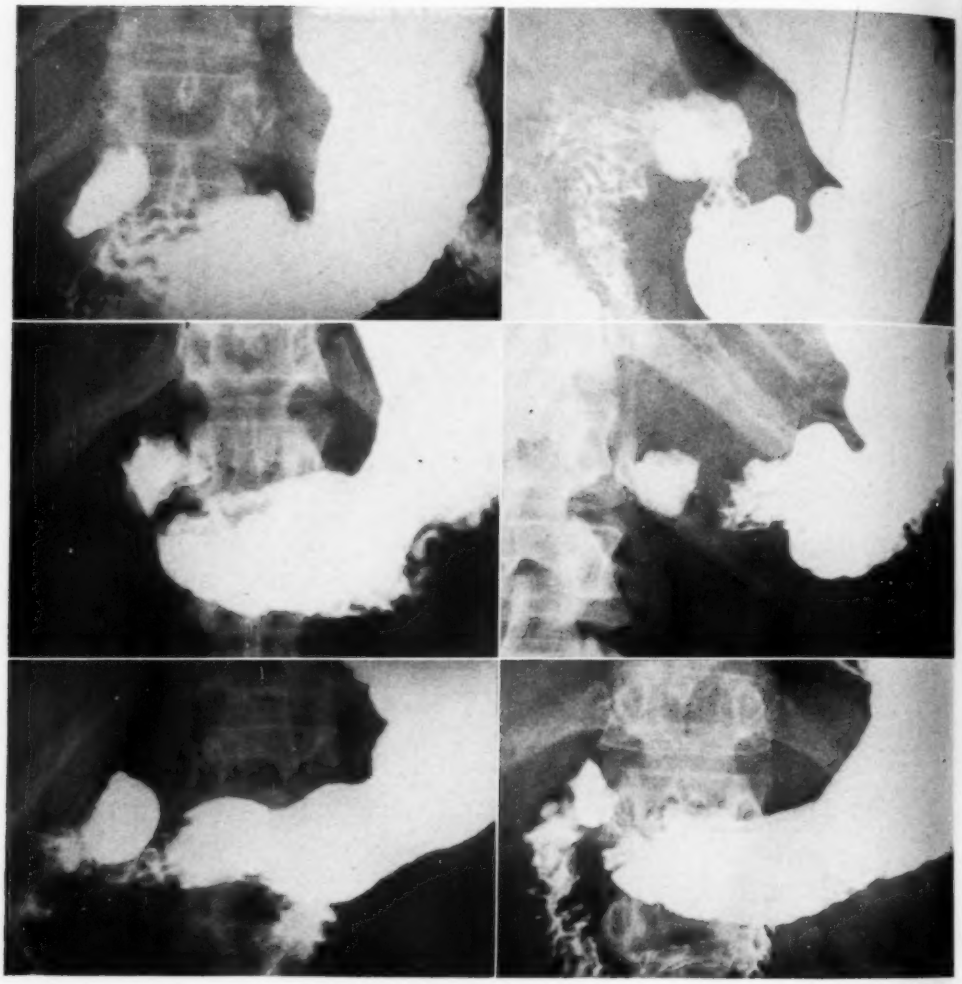

Plate III: Distal segment of the stomach in prepyloric local gastritis.

at the possibility of accurate x-ray demonstration of this condition. A recent article by Walk (11) supports this opinion. It is our contention that a properly conducted x-ray examination *can* give detailed information concerning the condition of the gastric mucosa. Our technic is as follows:

An aqueous suspension of 100 gm. of barium sulfate in a total volume of 240 c.c. is used. After preliminary fluoroscopic survey of the thorax and abdomen, the patient is instructed to take a single swallow of the barium suspension, "about a tablespoonful." The entry of the opaque material into the stomach and its subsequent descent are studied with particular care to ascertain the presence or absence of non-opaque gastric fluid. Manual pressure is exerted over the most dependent portion of the stomach, and the barium suspension is gently spread throughout the accessible portions of the stomach, a trace being forced through the pylorus if possible.

The mucosal folds within the stomach and duodenum are studied in detail, with spot-films as required to record and amplify the fluoroscopic observations. These are

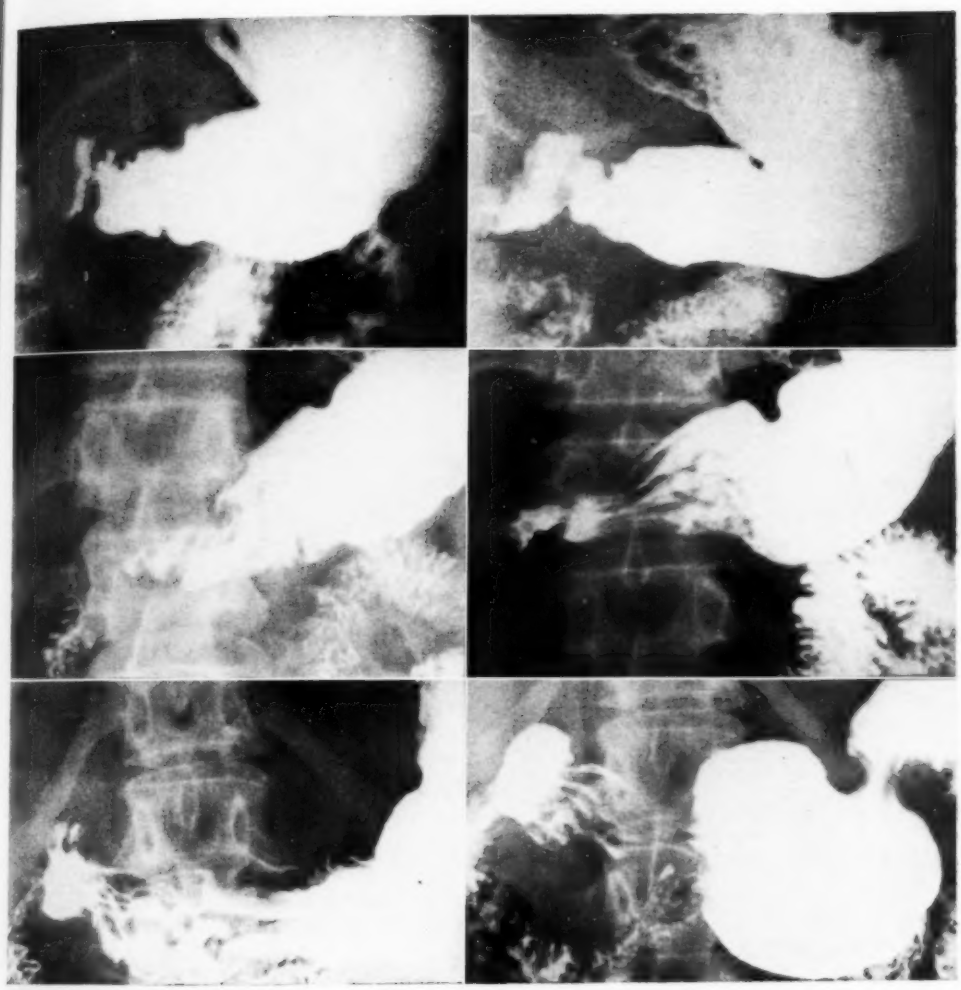

Plate IV: Prepyloric local gastritis, before and after treatment. Roentgenograms on the left are those of the initial examination in each case, while those on the right show the appearance after remission of symptoms. The interval is six weeks in each case.

generally made with the aid of an aluminum pressure cone. Only after this procedure has been carried to completion is the stomach filled with the remainder of the 240 c.c. meal.

Observation is then carried out with the patient erect, in frontal and oblique projections. Further fluoroscopic examination is done in the prone, prone right anterior oblique, and right lateral decubitus positions. Routine radiography is conducted in the prone and prone oblique positions, with such additional views as seem indicated in the individual case. Four-hour observation is also routine.

Our criteria of prepyloric local gastritis are (*a*) the presence of an initial gastric fluid residue ("hypersecretion"); (*b*) visible prepyloric mucosal deformity, as hereinafter described and illustrated; (*c*) localized prepyloric tenderness on deep pressure; (*d*) spasm along the distal portion of the lesser curvature, with or without pylorospasm; (*e*) interference with normal peristalsis and "antral systole." The presence of any four of these criteria is

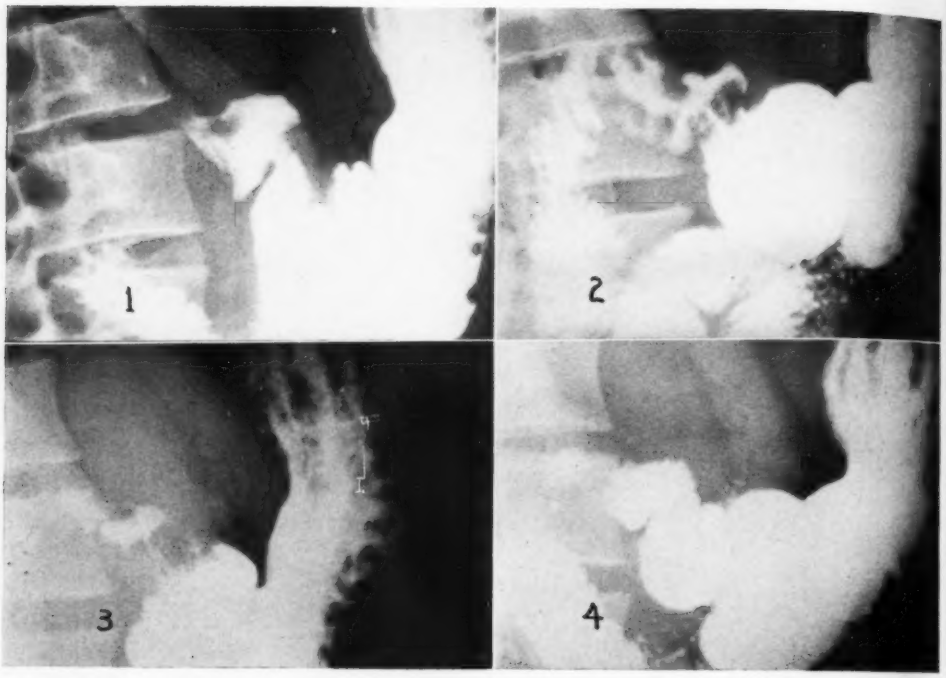

Plate V. Serial examinations (Case A. K.).
1. Initial examination, Dec. 16, 1947. Patient refused treatment after this date because of temporary remission of symptoms.
2. Feb. 17, 1948. Severe prepyloric local gastritis, duodenal ulcer.
3. April 6, 1948. Both the prepyloric local gastritis and the duodenal ulcer are healing.
4. July 19, 1948. The patient is asymptomatic and there is no radiologic evidence of disease. The fluoroscopic examination of this date and other roentgenograms demonstrated an apparently normal stomach and duodenum.

sufficient for diagnosis if mucosal deformity and local tenderness are among those found.

The characteristics of the deformed prepyloric mucosal folds can be manifold. There is almost invariably a thickening of the individual folds, with varying degrees of distortion of the pattern which they form. The changes are generally most marked along the lesser curvature, in its distal portion. The swollen folds may project through the pylorus to form filling defects in the base of the duodenal bulb. In several of our cases the changes, though localized, have been of such an extreme degree as to excite the suspicion of neoplasia; the stiffening of the gastric wall is, however, somewhat less than is usually seen in carcinoma, and the subsequent dramatic disappearance of the lesions under therapy has justified our diagnosis.

The series of roentgenograms in Plate IV demonstrates the healing of this disease under conservative medical management.

DISCUSSION

The course of several of these interesting cases has led us to speculate that prepyloric local gastritis may in numerous instances be the precursor of gastric or duodenal peptic ulcer. It has seemed remarkable that the roentgen findings of prepyloric local gastritis persist after the relief of symptoms, and after the apparent healing of gastric and duodenal ulcer craters. In such cases the recurrence rate has been high when the patient discontinued therapy before all radiologic signs of disease had disappeared. Plate V demonstrates a case in which a duodenal ulcer developed after prepyloric local gastritis was identified. Plate VI

shows a recurrence in a patient who failed to follow advice.

TREATMENT

Our fundamental rule in these cases is to give the gastric mucosa as much rest as possible. To this end, a bland diet is instituted, with "Amphojel" (aluminum hydroxide gel, U.S.P.), as in a peptic ulcer regime. The patient is urged to discontinue the use of alcohol, tobacco, and coffee. Atropine or belladonna is used in moderate dosage, with phenobarbital where its use seems desirable. "Pyribenzamine" (tripelennamine hydrochloride), 50 mg. q.i.d., is given for the first two weeks, after which the dosage is continued or reduced according to the indications in the individual case. This drug seems nearly specific for the disease and has alleviated symptoms even in cases in which co-operation has been poor.

The underlying psychodynamic factor is approached by discussion with the patient of the mechanism of the disease and the malevolent effects of tension and worry. When the patient can be induced to discuss his personal problems freely with his physician, an early and complete remission of symptoms can be expected.

We endeavor to emphasize to the patient the fact that he has a "six months disease" at the very least. We have found roentgen evidence of complete remission in most instances at from three to four months after the institution of therapy, but we believe that an additional period of dietary and pharmacologic supervision may diminish the recurrence rate.

SUMMARY AND CONCLUSION

Prepyloric local gastritis is a disorder manifested by postprandial epigastric distress and radiologic abnormalities of the distal segment of the stomach. The anamnestic and radiographic characteristics of the disease are discussed. We believe this condition to be a precursor of gastric and duodenal peptic ulcer in many instances. Its treatment is simple and effective.

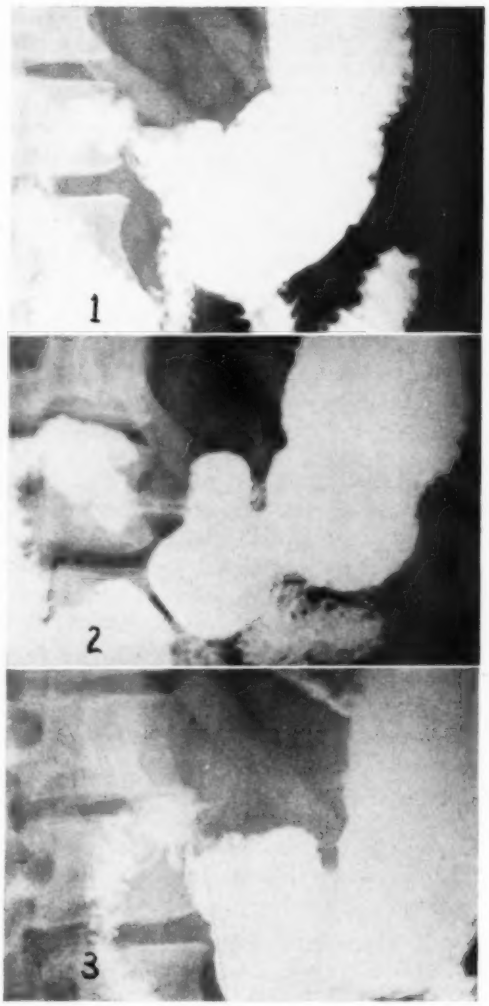

Plate VI: Serial examinations (Case R. S.).
1. Initial examination, Jan. 14, 1947.
2. Feb. 11, 1947. The patient has been free from distress for three weeks. The fluoroscopic examination and the other roentgenograms made on this date show no gastric or duodenal abnormalities.
3. March 24, 1947. The patient discontinued treatment, against advice, after the previous examination. He now exhibits a recurrence of prepyloric local gastritis.

The recognition and care of this condition will diminish greatly the number of undiagnosed dyspepsias in general medical practice.

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REFERENCES

1. FORSSELL, G.: Studies of the Mechanism of Movement of the Mucous Membrane of the Digestive Tract Am. J. Roentgenol. **10:** 87–104, February 1923.
2. RIVERS, A. B.: Clinical Study of Duodenitis, Gastritis, and Gastrojejunitis. Ann. Int. Med. **4:** 1265–1281, April 1931.
3. KIRKLIN, B. R.: Duodenitis and Its Roentgenologic Characteristics. Am. J. Roentgenol. **31:** 581–587, May 1934.
4. GOLDEN, R.: Antral Gastritis and Spasm. J.A.M.A. **109:** 1497–1500, Nov. 6, 1937.
5. WOLF, S., AND WOLFF, H. G.: Gastric Mucosa, "Gastritis," and Ulcer. Am. J. Digest. Dis. **10:** 23–28, January 1943.
6. SPRIGGS, E. I., AND MARXER, O. A.: Polyps of Stomach and Polypoid Gastritis. Quart. J. Med. **12:** 1–60, January 1943.
7. CAVE, E. H. P.: Value of Radiology in Diagnosis of Gastritis and Duodenitis. M. Press **212:** 268–271, Oct. 25, 1944.
8. VAUGHAN, W. W.: Antral Gastritis: Roentgenologic and Gastroscopic Findings. Radiology **44:** 531–54, June 1945.
9. ARENDT, J.: Chronic Hypertrophic Antrum Gastritis. Ann. Surg. **122:** 235–252, August 1945.
10. PORTIS, S. A.: Gastroenterological Aspects of Psychosomatic Medicine. Rev. Gastroenterol. **14:** 409–419, June 1947.
11. WALK, L.: Roentgen Signs of Gastritis. Am. J. Roentgenol. **60:** 77–79, July 1948.

SUMARIO

Gastritis Local Prepilórica

La gastritis local prepilórica es un trastorno que se traduce por malestar epigástrico postprandial y anomalías radiológicas del segmento distal del estómago, comprendiendo estas últimas: presencia inicial de un residuo de jugo gástrico; deformidad visible de la mucosa prepilórica (espesamiento de los pliegues de la mucosa con mayor o menor alteración del patrón que forman); espasmo a lo largo de la porción distal de la curvatura menor, con o sin pilorcespasmo; obstaculización del peristaltismo normal y sístole antral. Para el diagnóstico es indispensable la presencia de hiperestesia local y de deformidades de la mucosa.

Este estado parece ser en muchos casos precursor de úlcera gástrica o duodenal. El tratamiento es sencillo y eficaz, consistiendo en la medicación apropiada, la regulación dietética y la atención a los subyacentes factores psicodinámicos. Con el reconocimiento y cuidado del estado disminuirá considerablemente el número de dispepsias no diagnosticadas en la práctica médica general.

Las observaciones aquí anotadas se hicieron en una serie de 1,519 exámenes de la porción superior del aparato gastrointestinal en 1,289 enfermos, incluso 285 casos de gastritis local prepilórica.

Full Cycle Angiocardiography[1]

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ALTHOUGH EARLIER work in this direction had been accomplished, it remained for Robb and Steinberg in 1938 (1) to present to American radiologists a safe and practicable method of visualizing the various chambers of the heart in man. Thanks to them, angiocardiography was available for clinical use when new developments in cardiac surgery (2) emphasized the need for more accurate analysis of cardiovascular anomalies in prospective patients. The basic method described by these investigators has been widely used during the past decade. In many instances valuable anatomic information otherwise unobtainable has been provided in spectacular fashion. Sometimes, particularly in cases of congenital cardiac lesions, angiocardiographic examination has yielded disappointingly unsatisfactory results. Failure to identify adequately the nature of developmental anomalies involving the heart and great vessels is often traceable to shortcomings in existing apparatus employed for angiocardiographic examination.

The rapid advances in the field of cardiac and vascular surgery which the past few years have witnessed call for the development of radiologic methods which are capable of yielding precise information regarding detailed cardiovascular anatomy and circulatory physiology. Angiocardiography, if fully exploited, should be able to provide far greater diagnostic assistance than is now the case. Any specialized device designed for use in the radiographic study of cardiac circulation must meet four basic requirements of performance if the potential value of such examination is to be fully realized.

1. Individual exposures must be sufficiently brief to stop effectively the movement of cardiac walls and the advancing column of opacified blood. Exposures as rapid as one-sixtieth of a second are desirable; exposures which exceed one-twentieth of a second approach the allowable maximum.

2. Ability to provide multiple exposures in extremely rapid sequence is necessary if the entire cycle of cardiac circulation is to be observed. Within the beating heart, events occur so rapidly that an exposure rate of four per second may well be considered to be a minimum requirement.

3. Roentgenographic images must be brilliant and clearly detailed if they are to be interpreted with the degree of accuracy which examination of this type requires.

4. Apparatus used for angiocardiography must be automatic and dependable in operation. A rapid burst of automatically timed and spaced exposures is required to record the fleeting circulation of the opaque mass from vena cava to descending aorta.

Advantageous but less important features to be sought in addition to the basic requirements are easy mobility and reasonable cost. Unless in a given x-ray department it is possible to establish a separate room for highly specialized procedures, a room which need not be counted upon for the daily clinical activities of the division, it is helpful to use an angiocardiographic device which can be wheeled into a room equipped with high-energy generating equipment and as quickly wheeled out when study of the circulation through the patient's heart has been completed. Extremely high cost of any specialized x-ray accessory greatly limits its wide application, thus hampering and delaying desirable development.

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Today, in the laboratories throughout the United States where angiocardiography is being employed, apparatus of three general types is being used. One type of device depends upon the use of cut film in standard or special cassettes equipped with high-speed intensifying screens. Cassettes are shifted automatically from a lead-protected magazine into position for exposure and in the same fashion are removed from the exposure field and transported into a lead protected chamber after exposure (3–5). In general, instruments of this sort do not attain the frequency of exposure necessary to record in dependable fashion all of the important events of cardiac circulation.

The photofluorographic method has been adapted to the study of cardiac circulation with some success (6). The principal difficulties inherent in this type of apparatus are: some loss in roentgenographic detail; the necessity for greater exposure of the patient than is required when direct filming methods are used; and distinct limitation in the matter of frequency of exposure.

Recently magazine cameras designed for aerial photography have been adapted commercially to the needs of angiocardiography. Strip film is fed into position between intensifying screens where successive direct exposures of 9 1/2 × 9 1/2 inches are made. Thus far the manufacturer has been unable to offer greater frequency of exposure than two per second. The cost of this type of equipment is extremely high at the moment.

The Cinex camera designed by Dr. Hans Jarre and described by him in 1929 (7, 8) possesses many of the features which are desirable for angiocardiography. Several years ago Dr. Jarre kindly loaned his camera to us, and with it some attempts to study pulmonary circulation in experimental animals were carried out prior to

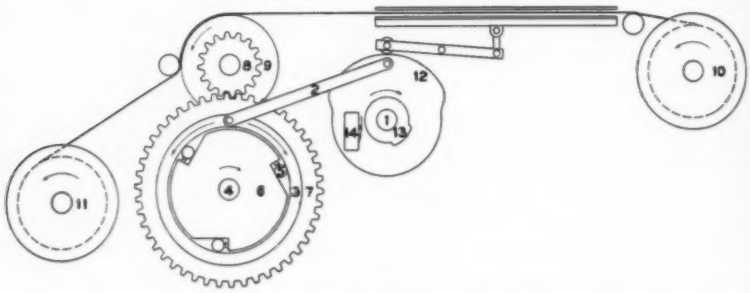

Fig. 1. Diagrammatic sketch of angiocardiographic camera: Drive shaft (1) is driven by a motor through a variable speed reducer allowing selection of speeds from 1 to 6 per second. An eccentric arm (2), operating from the drive shaft imparts a 120° oscillating motion to the outer ring (3) of the free-wheeling clutch. The outer ring floats freely on the shaft (4) and when turning clockwise causes the jamming pins (5) to jam between the opposing surfaces of the outer ring and the inner ring (6), thereby driving the inner ring. During counter-clockwise rotation of the outer ring, the jamming pins ride free between the opposing surfaces and hence impart no motion to the inner ring. Both the inner ring and the large gear (7) are keyed to the same shaft. The gear ratio between the large gear and the small gear (8) and the circumference of the film metering drum (9) are so designed that the intermittent 120° rotary motion of the large gear will meter the desired length of film over the metering drum. Roll film supplied from the storage spool (10) and passing between intensifying screens in the exposure frame is thereby metered in equal lengths. It is then wound on a take-up spool (11) which is driven through a simple slip clutch (not shown). The screen cam (12) on the drive shaft lowers the back screen by means of a lever arm, freeing the film during the film-moving portion of the cycle. Screen contact during the exposure portion of the cycle is maintained by spring tension. The radiographic exposure is synchronized with the exposure portion of the camera cycle by means of the timing cam (13) which closes the normally open micro-switch (14) immediately after the film comes to rest. The leads from the micro-switch are connected to an ordinary female lamp plug which is installed in the radiographic control panel and wired in parallel to the push-button circuit.

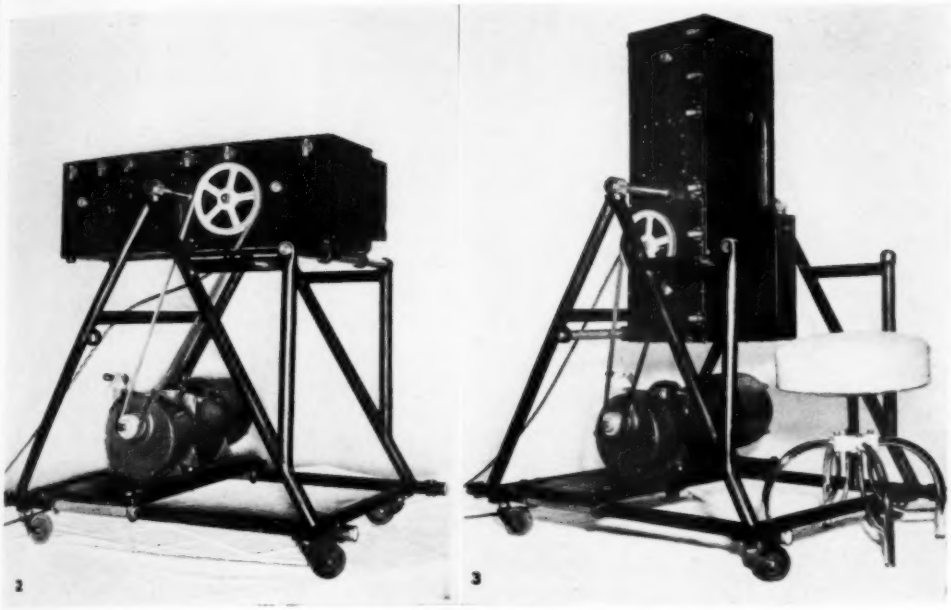

Fig. 2. Modified cinex-camera, motor-driven, with carriage. Horizontal position.
Fig. 3. Camera arranged in vertical position.

July 1946, when the instrument was returned to Dr. Jarre. In our machine shop we constructed a camera similar to the Cinex for use with 8-inch duplitized strip film with motor drive supplanting the original hand-crank operation. Very quickly it was found necessary to redesign the power transmission mechanism completely to allow smooth and dependable operation. The revised mechanism, diagrammed in Figure 1, has performed very satisfactorily since November 1947 in carrying out something over fifty angiocardiographic examinations in man.

A sturdy wheeled carriage was built to support the camera in either the horizontal or upright position (Figs. 2 and 3). The front surface of the camera consists of three hinged doors which may be opened for loading and unloading. These latch tightly to exclude light when the instrument is in operation (Fig. 4). The two end doors, which cover the supply and take-up film spools, are heavily leaded to exclude x-rays. The center door carries one of the two 8 × 10-inch high-speed intensifying screens between which film is exposed. The camera can be loaded with a spool of

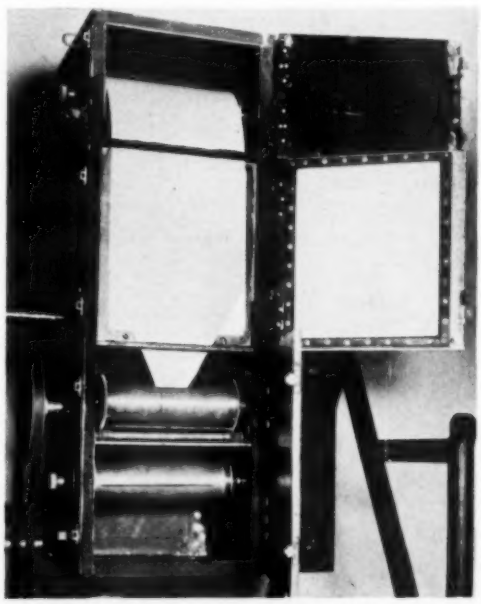

Fig. 4. Camera with its three hinged doors open.

x-ray film 8 inches wide and 50 feet or more in length.

Although the instrument is capable of advancing film at the rate of five frames per second, it has not been possible to date to take full advantage of this feature. The 500-ma. energizing apparatus installed in the radiographic room where the camera is used is equipped with a mechanical impulse timer which was not designed for rapidly repeated exposures. The contact switch will not reset itself rapidly enough to permit exposures more frequently than two and one-half per second. Even with this limitation, which has not yet been overcome, consecutive exposures at half-second intervals from the moment of injection of the opaque mass until its dissipation in the systemic arterial system has provided a wealth of detailed information regarding circulatory events.

The desirability of showing the complete cycle of circulation from peripheral vein to aortic subdivisions is illustrated in Figure 5. In this instance the patient presented clinical indications of aortic coarctation. From an uninterrupted sequence of 28 exposures made at half-second intervals, nine frames have been selected which show the highlights of the entire series. Films are sequentially numbered from the entry of the injection mass into the superior vena cava, the first film with opaque blood in the superior vena cava being number 1. Along the top row, Exposures 2, 3, and 5, made one-half, one, and two seconds after vena caval fill, show the progress of opaque material from the right auricle through the right ventricle and into the finer branches of the pulmonary arteries. The middle row, Exposures 8, 10, and 13, made three and a half, four and a half, and six seconds after vena caval fill, show virtual disappearance of opaque material when the injection mass is almost completely confined to the pulmonary capillary bed and then the spectacular return of the opaque material to the pulmonary veins, with ultimate filling of the left auricle and ventricle and partial escape into the ascending limb of the aorta.

Illustrations in the bottom row, Exposures 14, 15, and 16, show rapidly changing events at half-second intervals as the left ventricle discharges into the aorta and its branches. It is of considerable interest to note that, unless one were obtaining exposures at closely spaced intervals, the very clear demonstration of the aortic constriction so nicely seen in Exposure number 15 would have been obtained only by the merest chance. A half second earlier and a half second later the point of constriction is seen with difficulty. Attention is called to the fact that, although the innominate and left common carotid arteries are clearly visible, the subclavian cannot be seen. In this particular patient, upper extremity hypertension characteristic of aortic coarctation is confined to the right arm, where the systolic pressure was read at 165 mm. Hg, in contrast to 110 mm. Hg on the left. It is noteworthy that information of this character can be of the utmost value to the thoracic surgeons who contemplate carrying out the Blalock procedure in a patient with the tetralogy of Fallot or repair of aortic coarctation.

It is gratifying to be able in this particular patient to observe in excellent detail: the configuration of the right ventricular outflow tract (Exposure 5); the right surface of the interventricular septum (Exposure 3); the period of pulmonary capillary fill (Exposure 8); filling of pulmonary veins (Exposure 10); the left margin of the interventricular septum (Exposure 13) with beginning fill of the ascending aorta; and the all-important identification of coarctation site (Exposure 15). This very achievement arouses the great desire to observe with even greater detail the sequence of events instant by instant during cardiac circulation. For example, in the brief space of one-half second, the elapsed time between Exposure 2 and Exposure 3, extensive ventricular emptying has occurred, with virtually complete filling of the pulmonary arteries and their minor branches. Only by good fortune did Exposure 5 again catch the right ventricle in late systole, thus exposing

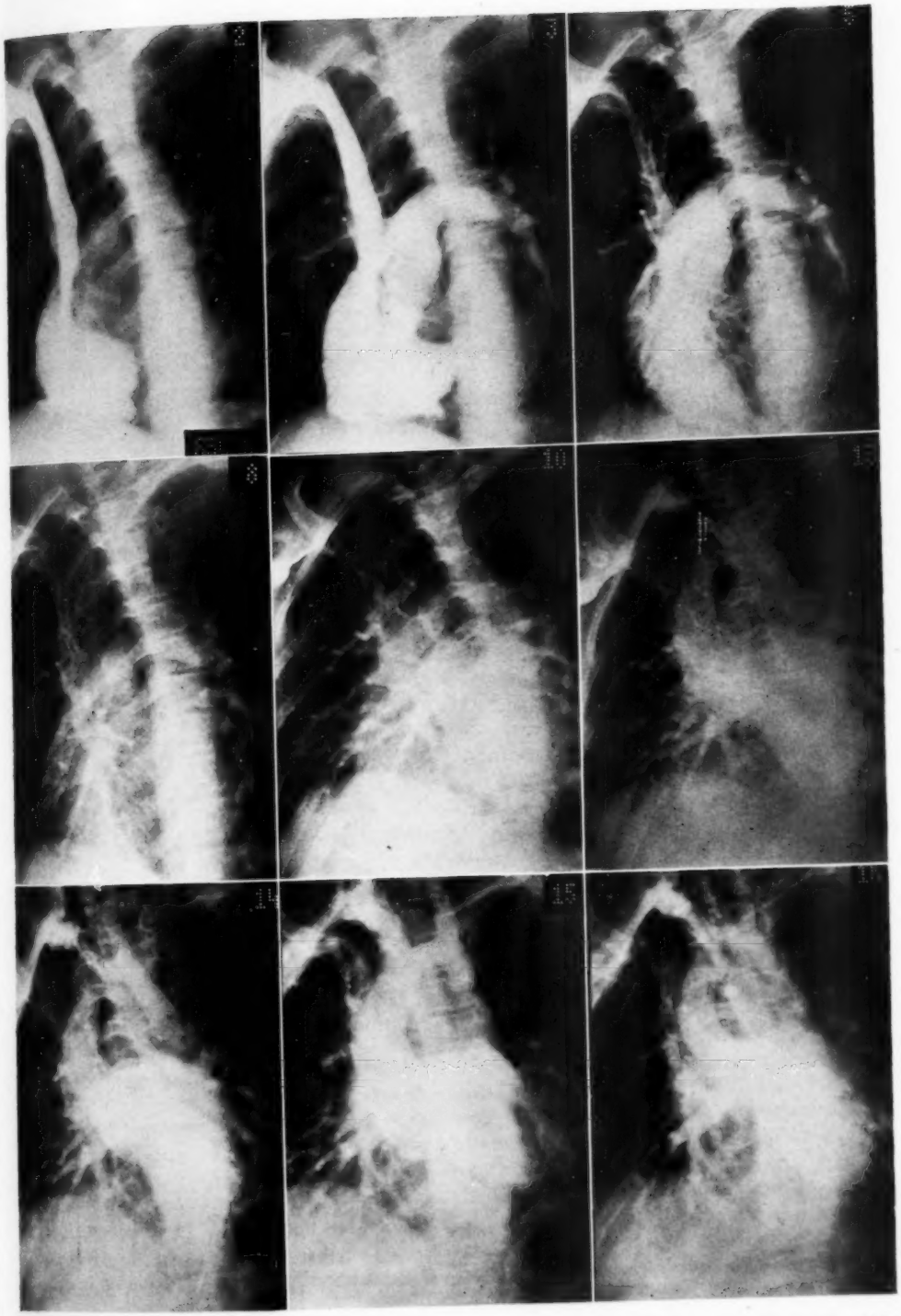

Fig. 5. Selected exposures from recorded circulation in a patient with aortic coarctation. Female, age 16, weight 75 lb.; 35 c.c. 70 per cent diodrast; 300 ma., 92 kv., 1/20 second with wafer grid. Exposure rate two per second.

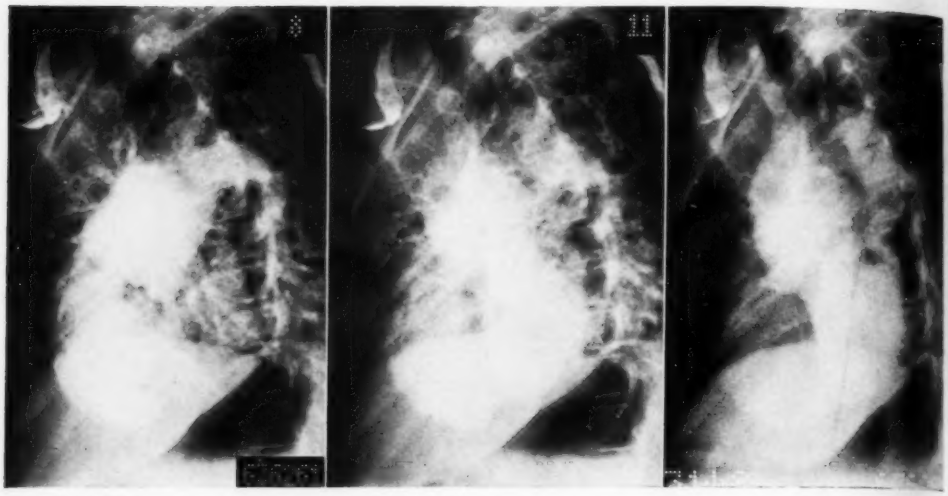

Fig. 6. Interauricular septal defect. Three exposures from complete series, showing continuing opacity of right heart. Female, age 8, weight 40 lb.; 30 c.c. 70 per cent diodrast; 300 ma., 90 kv., 1/20 second with water grid. Exposure rate two per second.

the right ventricular outflow tract to good advantage. The considerable variation in configuration of the left ventricle as shown in Exposures 13, 14, 15, and 16 causes one to wish for enough more exposures to record the sequential changes in shape and size which occur throughout the cycle of ventricular contraction.

Another argument for multiple sequential exposures to show the entire story of cardiac circulation is illustrated by Figure 6, an examination from which three exposures have been selected. This patient was thought, on the basis of clinical findings, to have uncomplicated interauricular septal defect. The interauricular septal defect has permitted opaque material to re-enter the right heart from the left auricle, showing as simultaneous filling of the right and left ventricles (Exposure 11) five seconds after injection. One could scarcely be expected to plan in advance the optimum instant of exposure to show this state of affairs. In Exposure 8 there appears to be stenosis of the right ventricular outflow tract with associated dilatation of the pulmonary artery peripheral to this point. It is entirely possible, however, that as seen at this particular instant, when systole appears to be about over, the caliber of the outflow tract as shown may not represent the true state of affairs. Actually large pulsations were observed in the pulmonary artery.

The imperative need for more complete documentation of the events of cardiac circulation is spectacularly demonstrated by Figure 7. This patient has situs inversus of abdominal organs and levocardia. The three exposures here reproduced, made at one-half, one, and one and one-half seconds after injection, show that incoming opaque material first seen in the transposed right auricle has filled all cardiac chambers in the interval of one-half second without providing the highly desirable information as to the manner in which this was accomplished. Filling of the right-sided aorta, which has already begun in Exposure 3, can be followed into the abdominal segment one-half second later (Exposure 4). In this case the opaque mass has passed through the heart so rapidly that very few circulatory details have been recorded. In another instance of transposed viscera with levocardia (Figure 8), in the short space of three and one-half seconds (Exposures 2–7) the important events of circulation through the grossly anomalous heart and great

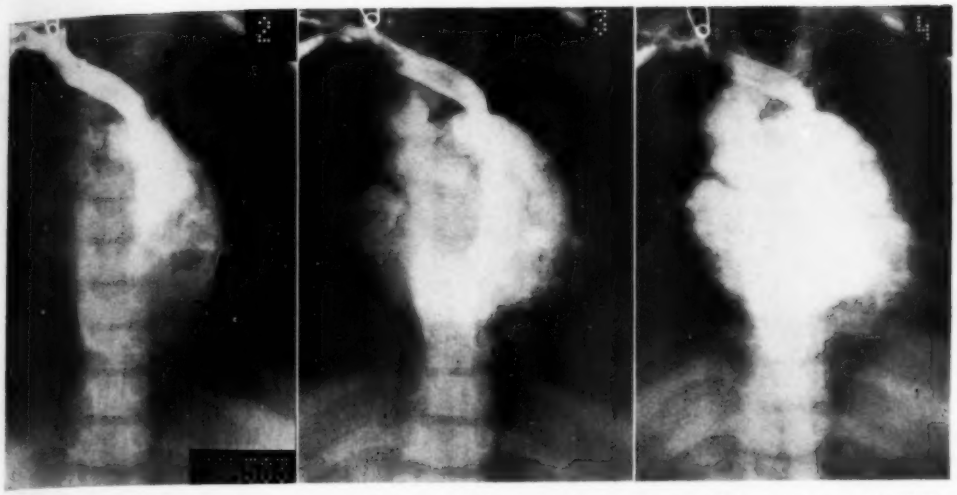

Fig. 7. Transposition anomaly showing confusingly rapid filling of all chambers. Male age 4, weight, 40 lb.; 20 c.c. 70 per cent diodrast; 400 ma., 92 kv., 1/30 second with wafer grid. Exposure rate two per second.

Fig. 8. Transposition anomaly showing fleeting opacity of all cardiac chambers. Female, 3 1/2 mo.; 10 c.c. 75 per cent neo-iopax; 300 ma., 58 kv., 1/40 second, no grid. Exposure rate two per second.

vessels are virtually complete. In Exposure 2, two superior venae cavae are seen about to discharge their contents into the heart. One second later (Exposure 4), well before emptying of the venae cavae has occurred, opaque material has spread through the heart and out into the proximal aorta. One and one-half seconds later, chambers within the heart are almost indistinguishable and opaque filling of the abdominal aorta has occurred.

It is not always simple to show the right ventricular outflow tract to advantage. On the other hand, information regarding the caliber of this tract is most important in determining whether or not the establishment of a surgical communication between systemic and pulmonary circuits is desirable. Figure 9 illustrates the normal degree of caliber change one may expect to see if this canal can be observed in all phases of the cardiac cycle. The width of

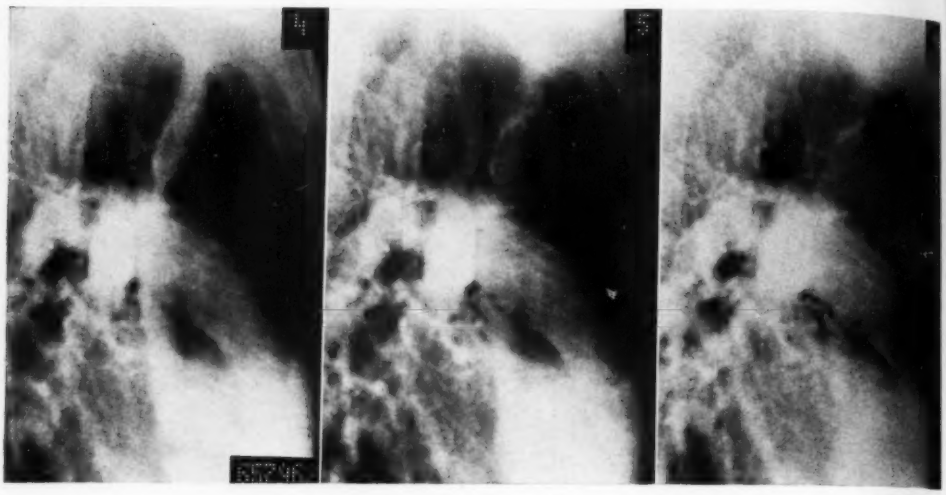

Fig. 9. Aortic coarctation, showing normal variability of right ventricular outflow tract in three successive exposures. Male, age 38, weight 165 lb.; 50 c.c. 75 per cent neo-iopax; 300 ma., 95 kv., 1/15 second with wafer grid. Exposure rate two per second.

the tract, shown here in left lateral projection, becomes progressively narrower in Exposures 4, 5, and 6, made as usual at one-half second intervals. It would have been most helpful had the instant of these three exposures been recorded on a simultaneous electrocardiographic tracing. On the basis of Exposure 6 alone, one might have considered reporting abnormal narrowing of the infundibulum of the right ventricle.

An interesting and provocative situation is illustrated in Figure 10, exposures selected from a case of uncomplicated interventricular septal defect. Seen in right lateral projection the right pulmonary artery is recorded in what might be called optical section. It casts a dense disk-like shadow which varies sharply in diameter from exposure to exposure. Here again, identification of the instant of exposure upon an electrocardiographic tracing, together with exposures at much more frequent intervals, would have supplied interesting information regarding the amplitude of pulsation of this vessel.

The full extent to which roentgenographic methods can be exploited to study the complex procession of events occurring during the flow of blood through the chambers of the heart and its great vessels has not yet been achieved. There are no insurmountable problems involved, however. A new model of our camera is now under construction which will provide direct exposures eleven inches square to minimize present difficulties in the matter of accurate positioning of patients. This camera will be capable of recording exposures at the rate of six per second. The completed instrument will be considerably lighter than its prototype and will be designed with removable lightproof magazines to obviate the necessity of transporting the entire instrument into a darkroom for loading and unloading.

To utilize the full capabilities of the new camera an electronic timer, currently being developed commercially, will be employed to overcome present difficulties with mechanical timers. Initial information from the manufacturer indicates that this new timer will be able to deliver eight 1/30th-second exposures in one second.

We are also adapting a direct writing, two-channel oscillograph with appropriate amplifying device to integrate sequential roentgenographic exposures with continuously recorded electrocardiographic tracings. It is our present plan to build a

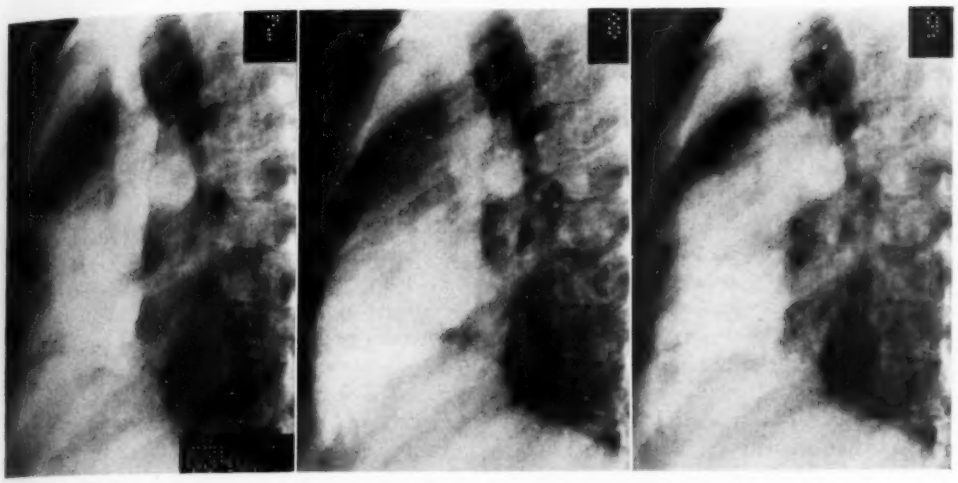

Fig. 10. Uncomplicated interventricular septal defect. Three successive exposures show caliber changes in right pulmonary artery. Female, age 9, weight 40 lb.; 15 c.c. 70 per cent diodrast; 200 ma., 82 kv., 1/10 second with wafer grid. Exposure rate two per second.

control panel through which the camera and the electrocardiographic recording device can be operated by one individual.

SUMMARY

Much of interest and value can be learned from cardiac angiography when the full cycle of circulatory events during the passage of blood from the vena cava to the aorta is recorded. Existing apparatus which can be operated at the rate of two exposures per second is a long step in this direction. Results obtained with such apparatus whet the appetite for an instrument which is capable of providing much more frequent sequential exposures—exposures accurately related to events recorded by simultaneous electrocardiography.

The design of an instrument is described which promises to permit much more extensive exploration of intrathoracic circulation by means of the angiocardiographic method.

REFERENCES

1. ROBB, G. P., AND STEINBERG, I: A Practical Method of Visualization of the Chambers of the Heart, the Pulmonary Circulation, and the Great Blood Vessels in Man. J. Clin. Investigation **17:** 507, July 1938.

2. BLALOCK, A., AND TAUSSIG, H. B.: Surgical Treatment of Malformations of the Heart in Which There Is Pulmonary Stenosis or Pulmonary Atresia. J. A. M. A. **128:** 189–202, May 19, 1945.

3. SUSSMAN, M. L., STEINBERG, M. F., AND GRISHMAN, A.: A Rapid Film Changer for Use in Contrast Angiocardiography. Radiology **38:** 232–233, February 1942.

4. SCHWARZCHILD, M. M.: A Multiple Cassette Changer for Angiocardiography: A Device for Rapid Serial Radiography. Radiology **40:** 72–74, January 1943.

5. TAYLOR, H. K.: An Apparatus for Automatic Multiple Exposures. Radiology **52:** 107–111, January 1949.

6. TEMPLE, H. L., STEINBERG, I., AND DOTTER, C. T.: Angiocardiography Utilizing Photoroentgen Apparatus with a Rapid Film Changer. Am. J. Roentgenol. **60:** 646–649, November 1948.

7. JARRE, H. A.: The Cinex-Camera. Grace Hospital Bulletin, July 1929.

8. JARRE, H. A.: Roentgenologic Studies on Physiologic Motor Phenomena (with the Aid of the Cinex-Camera). Radiology **15:** 377–393, September 1930.

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(*For Spanish summary, see following page*)

SUMARIO

Angiocardiografía de Ciclo Completo

Puede averiguarse mucho de interés y valor con la cardioangiografía cuando se registra todo el ciclo de acontecimientos circulatorios durante el paso de la sangre de la cava a la aorta. El aparato actual que puede funcionar a razón de dos exposiciones por segundo representa un adelanto considerable en ese sentido. Los resultados consequidos con dicho aparato excitan los deseos de obtener un instrumento capaz de facilitar exposiciones consecutivas mucho más frecuentes, exposiciones estas relacionadas exactamente con los hechos registrados por la electrocardiografía simultánea.

Descríbese el diseño de un instrumento en vías de construcción que, según parece, permitirá una exploración mucho más extensa de la circulación intratorácica con la técnica angiocardiográfica. El nuevo modelo suministrará exposiciones directas de 27.5 cm.^{2} para reducir al mínimo las actuales dificultades con respecto a la colocación exacta en posición de los enfermos. También podrá registrar exposiciones a razón de seis por segundo. Un cronografillo electrónico, ya en construcción para la venta, eliminará los obstáculos creados por los actuales cronógrafos mecánicos.

Roentgen Manifestations of Q Fever[1]

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Q FEVER HAS BEEN found to exist among the general population in and around Los Angeles, where a survey by the United States Public Health Service has shown it to be endemic (1, 18). Over 300 cases have been recognized in Southern California since the disease was first suspected in this region by Young (24) in May 1947. Slightly less than half of the patients were hospitalized during the acute phase of their illness, affording the authors an opportunity to study the roentgen manifestations. This report is based on a survey of the roentgenograms of the chest of 77 patients, selected on the basis of availability and completeness of records. Thirty-nine of this group were admitted to the Los Angeles County Hospital, the remainder to various other hospitals in the area.

HISTORICAL

Q fever was first described by Derrick (8) in Australia in 1937 following an outbreak of an obscure febrile illness among packing house workers. The rickettsia which was isolated was proved to be the causative organism of the diseases known as "nine-mile fever" in Montana and "Balkan grippe" in Southern Europe, as well as the "Q fever" of Australia. Outbreaks of Q fever occurred among Allied troops in the Mediterranean Area in 1944 and 1945 (22) and among troops returning to this country from that region (12). Q fever has also been reported from Panama (4) and Switzerland (13).

The first known naturally occurring outbreaks of Q fever in the United States took place in Amarillo, Texas, in March 1946, with 55 cases, including two deaths (17). A second outbreak of 36 cases occurred in Chicago in August 1946 (23). Both of these epidemics were explosive in onset, of short duration, and limited to persons handling cattle and sheep en route to or during slaughter. Lennette and Meiklejohn (19) reported two outbreaks of Q fever in Central and Northern California occurring between February and May 1948, with a total of more than 100 cases. In most cases there was a history of close contact with livestock, but in some no such history was obtainable.

ETIOLOGY

The causative agent of Q fever is a rickettsia which was first isolated by Burnet (3) in Australia and Davis and Cox (6) in the United States. The organism, *Coxiella burnetii* (Derrick) is a minute Gram-negative rod which is resistant to drying, chemicals, and heat (16). It passes through Berkefeld filters. It may be recovered from the blood stream of patients with Q fever and has been found in most of the organs at autopsy (2).

EPIDEMIOLOGY

The organism has its chief reservoir in cattle and has been found in more than half of the unpasteurized milk specimens tested in Los Angeles County (15). There is no person-to-person transmission of the disease. The incidence is highest among meat packers, laboratory workers studying *Coxiella burnetii*, and those working in or living near dairies. Nearly one-third of the patients in Los Angeles County denied any exposure to cattle or unpasteurized milk. The exact mode of transmission to man is unknown. One attack of Q fever confers immunity for an indefinite period of time. Relapses may occur during convalescence from the acute illness. There are no instances of a second attack even in

[1] From the Departments of Radiology and Medicine of the University of Southern California School of Medicine and Los Angeles County Hospital. Accepted for publication in December 1948.

laboratory or other workers who are repeatedly exposed.

HISTOPATHOLOGY

Little is known of the actual histopathology of Q fever. Eight deaths, including 3 in Southern California, are known to have occurred as a result of this disease (2, 8, 16, 17, 20). Only 2 autopsies have been reported. One of the fatalities occurred during a laboratory outbreak in the National Institute of Health (11, 14). Lillie, Perrin, and Armstrong (20), described, in part, a "diffuse consolidation of the lungs, with alveoli, bronchioles, and most bronchi filled with an exudate which is chiefly fibrinocellular.... The exudate is usually quite compact, with fibrin as the chief component. The cells are of small to moderate number in each alveolus, are usually enmeshed in fibrin and consist chiefly of lymphocytes, plasma cells, and large mononuclear cells; red blood cells are numerous in scattered alveoli but polymorphonuclears are few thruout.... A little fibrinocellular exudate on the pleura shows beginning organization..." The pathological changes in experimentally produced Q fever in monkeys and guinea-pigs are reported by the same authors as being entirely similar (20, 21).

CLINICAL FEATURES

The clinical features of the cases upon which this paper is based are being reported in detail elsewhere (7) but may be summarized as follows:

The age of the patients varied from three to seventy-five years. Eighty per cent were adult males. The most common symptom complex was that of a sudden onset of fever, chilly sensations, malaise, anorexia, and severe headache, followed in a few days by a slight, hacking, nonproductive cough and mild pleuritic chest pain. Primary atypical pneumonia was the initial tentative diagnosis in approximately 50 per cent of the patients. A severe but transitory meningism occurred in approximately one-fourth of the cases. A third group was characterized by fever and a generalized constitutional reaction without clinical evidence of either pulmonary or meningeal involvement. Roentgen examination of the chest showed most of these patients to have pneumonia.

The patients were febrile, with temperatures ranging from 101 to 104° F., had a relative bradycardia, and appeared acutely ill. The fever lasted seven to fifteen days in 90 per cent of the cases, the average being ten days, and subsided by lysis. Chest pain and pulmonary physical findings, when present, usually corresponded in location to that of the pneumonia, as shown by the roentgenograms. Skin rash and palpable splenomegaly were very rare. An occasional patient had blood-streaked sputum. Severe headache, often frontal or retro-ocular, was usually the chief complaint.

Penicillin, sulfonamides, streptomycin, para-aminobenzoic acid, and roentgen irradiation had no demonstrable effect upon the duration or severity of the illness. Aureomycin was used in 1 case in this series with apparently favorable results.

Recovery was usually prompt and complete. Convalescence was occasionally prolonged, with complaints of anorexia, asthma, and mild malaise persisting for several months. There have been no complications directly attributable to Q fever. The general mortality rate is approximately 1 per cent, there being a total of 3 known deaths from the disease in Southern California. None of the fatal cases is included in this series.

LABORATORY FINDINGS

The white blood counts varied from a leukopenia to a mild leukocytosis, but were usually within normal limits. As a group, the leukocyte counts showed a mild polymorphonuclear leukocytosis and an absolute lymphocytopenia. There was no significant alteration in this blood picture during the acute illness or early convalescence. The erythrocyte sedimentation rate was elevated in every case, with a mean corrected rate of 30 mm. per hour by the Wintrobe method. It remained

elevated during at least the first two weeks of illness. Urinalyses were normal except for transient albuminuria during the acute febrile period. The spinal fluid was normal except for some increase in pressure and a slightly decreased chloride content. The Weil-Felix and cold agglutinin tests were negative. No false positive serologic tests for syphilis have been noted.

The complement-fixation test for Q fever was used as the basis for diagnosis in every case. The tests were performed by the laboratories of the National Institute of Health, Bethesda, Md., and the California State Department of Public Health, Sacramento, Calif. The minimal criterion for the diagnosis of Q fever was a single blood specimen with a titer of 1:32 or greater taken during convalescence from an acute febrile illness clinically compatible with the disease. One-half of the cases studied had a fourfold or greater rise in titer in successive blood specimens. Twenty-five showed a change in serial blood specimens from a negative to a positive reaction. The complement-fixation test becomes positive during the second week of illness.

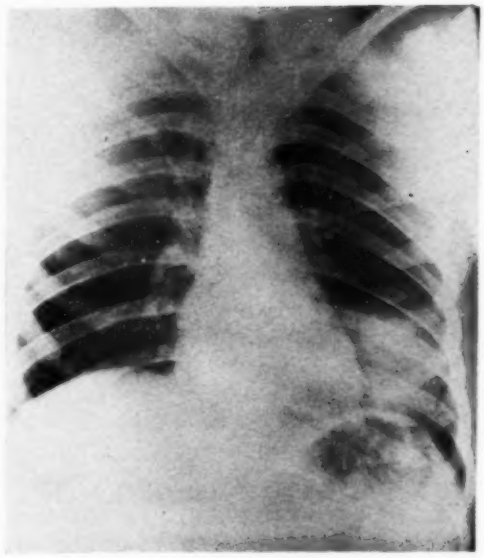

Fig. 1. H. C., age 19, Caucasian male; dairy farm worker. Sudden onset 3/28/48. Moderate malaise, headache, fever, and slight cough. Pleural pain in left chest. No upper respiratory symptoms. Admitted to hospital 4/3/48. Acutely ill. Dullness and râles at left base. Erythrocyte sedimentation rate 47 mm./hr. White blood cells 10,000 (polymorphonuclears 55%, lymphocytes 36%, monocytes 8%, eosinophils 1%). Maximum fever 104° F. Duration of fever twelve days. Q fever complement-fixation titer: 4/2/48 negative; 4/8/48 positive 1:64.

Roentgenogram made on seventh day of illness, 4/3/48. Segmental homogeneous consolidation, base of left lower lobe.

One day after the cessation of fever, partial resolution had occurred.

REVIEW OF PREVIOUSLY REPORTED ROENTGEN FINDINGS

In an outbreak of Q fever in the Mediterranean Area among American troops, Robbins and Ragan (22) reported 266 cases, in 78 of which there was no evidence of pneumonia. Radiographs were not obtained in all cases, however, so that the actual incidence of pneumonia is not known. The majority of those examined had single lesions; one had involvement of two lobes. The lower lobes were most frequently affected. Consolidation first appeared on the third or fourth day and in some cases as late as the sixth. Minimal pleural effusion was present in 4 of 51 patients. None had a large effusion. No correlation was noted between the clinical severity of the disease and the degree of pulmonary involvement.

Feinstein, Yesner, and Marks (12) reported 102 cases of Q fever in troops returning from Italy. The lesions persisted from a few days to several weeks, the duration averaging 10.8 days. There was pleural involvement in 10 cases. A number showed distinct small disseminated infiltrations. Some resembled atypical pneumonia, some tuberculosis (upper lobes), and others, where a complete bronchopulmonary segment was involved, bacterial pneumonia.

Irons and Hooper (17), reporting a series of 18 cases, described the pulmonary lesions as being frequently of the patchy type seen in bronchopneumonia, soft, diffuse, single or multiple, and lacking the uniform density of lobar pneumonia. Seldom was more than one lobe involved. Again there was no correlation between clinical severity and the degree of pulmonary involvement.

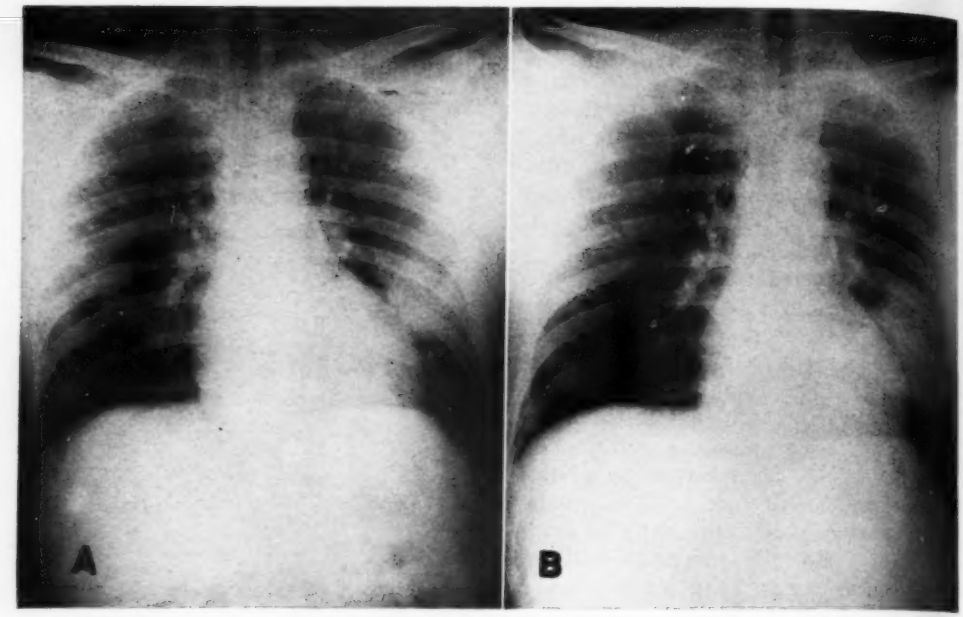

Fig. 2. G. T., age 28, Caucasian male; salesman. No history of exposure. Sudden onset 11/15/47. Moderate malaise, slight anorexia, weakness, myalgia, fever, chills, and severe frontal headache. No cough, pleural pain, or upper respiratory symptoms. Admitted to hospital 11/17/47. Acutely ill. Positive Kernig and slightly stiff neck. No pulmonary findings on physical examination. White blood cells 12,000 (polymorphonuclears 84%, lymphocytes 16%). Erythrocyte sedimentation rate 38 mm./hr. Maximum fever 105.4° F. Duration of fever nine days. Q fever complement-fixation titer: 11/17/47 negative; 11/28/47 positive 1:32.

A. Roentgenogram made on fourth day of illness, 11/18/47. Homogeneous, uniform area of consolidation in the left lower lobe. Scattered parenchymal calcifications.

B. Roentgenogram made on sixth day of illness, 11/20/47. Area of consolidation has increased by direct extension.

An outbreak of Q fever occurred among laboratory personnel of Fort Bragg, N. C. (5). Fourteen out of 16 patients had roentgen evidence of pneumonia; 10 had involvement of a single lobe; 4 of multiple lobes. The lower lobes (right and left) were involved 14 times, the left upper 3 times, the right upper 4 times, and the right middle lobe once. In 12 cases the pneumonia was evident during the first six days and in 8 by the third day. The earliest lesions were seen in the peripheral portions of the lung. They tended to be circular, ground glass in appearance, and more dense in their centers. Increase in involvement occurred by direct extension. The hili were reported as being singularly free of involvement. Maximum infiltration was noted between the sixth and ninth days, at about the time that the temperature became normal. There was evidence of collapse in 2 cases. Resolution required from three to six weeks.

ROENTGEN FINDINGS IN PRESENT SERIES

Of the 77 cases presented, 12 (16 per cent) showed no roentgen changes which could be attributed to Q fever. The remainder, 65 cases, had pneumonic infiltrations of varying extent.

Segmental or lobular consolidation was noted 43 times and lobar consolidation 16. Mottled confluent infiltration occurred in 6 cases and scattered patchy infiltration in 3. A small solitary patch was seen twice as the only lesion and 3 times in conjunction with other lesions.

The lesions were rather evenly distributed between the right and left lungs, with the following frequency: right lower lobe, 20; right middle lobe, 10; right upper lobe, 10; left lower lobe, 27; left upper

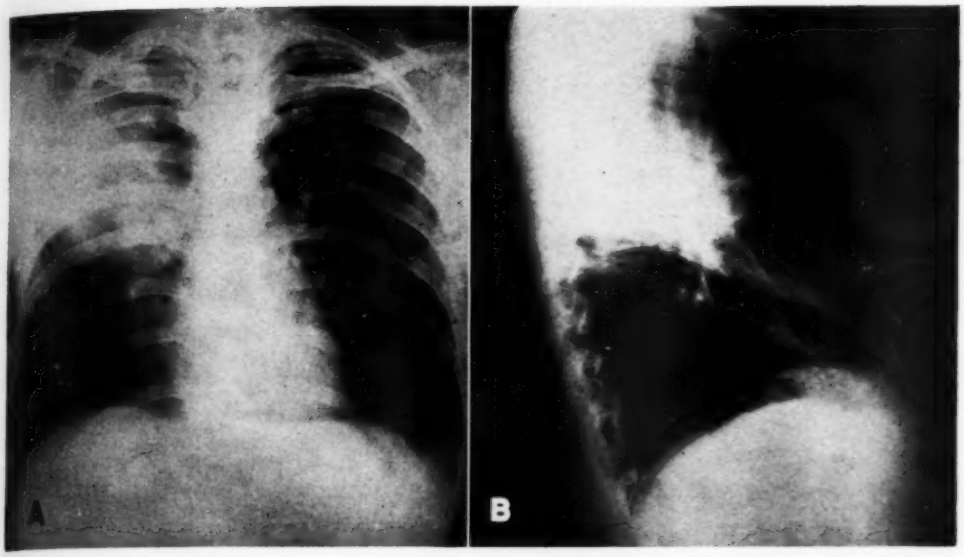

Fig. 3. R. O'L., age 33, Caucasian male; salesman. No history of exposure. Sudden onset 11/24/47. Moderate malaise, severe headache, chills, fever, stiff neck, sore throat, moderate dry cough. Pleural pain in right chest. Admitted to hospital 12/2/47. Acutely ill. Dullness and râles in right mid-lung field. White blood cells 7,800 (polymorphonuclears 88%, lymphocytes 10%, monocytes 1%, eosinophils 1%). Maximum fever 103.2° F. Duration of fever fourteen days. Erythrocyte sedimentation rate 21 mm./hr. Q fever complement-fixation titer: 12/11/47 positive 1:8; 1/22/48 positive 1:64.

A and B. Roentgenograms made on ninth day of illness (12/2/47). Dense, homogeneous consolidation in posterior basal segment of right upper lobe. Accentuation of interlobar fissures.

Fifteen days after the cessation of fever complete resolution had occurred.

lobe, 17. Eighty-three per cent of those with pneumonia had either segmental or lobar consolidations which were, therefore, the most characteristic lesions seen in this series. These were rather consistently homogeneous in appearance and accentuated towards the periphery of the lung fields. They varied in density from a diffuse cloudy consolidation to complete opacity.

Thirteen (24 per cent) of those with pneumonia had involvement bilaterally and of multiple lobes. Ten of these had either segmental or lobar consolidation of one lobe associated with similar lesions in one or more lobes in 6 instances, with mottled confluent infiltration in 2, with scattered patchy infiltration in 1, and with a solitary patch in 1. In 5 cases there was mottled confluent involvement of more than one lobe and in 1 sparsely scattered patches of infiltration throughout both lung fields. More than one radiographic study of the chest was available in 6 of these patients but in only 1 were there new areas of pneumonia, other than by direct extension, subsequent to the initial examination. The migratory infiltrations so often seen in atypical and virus pneumonias appear not to be a feature of Q fever.

Minimal amounts of pleural fluid were noted in 17 instances; small amounts of pleural fluid in 3 and in 1 a moderate amount of fluid. In none was there a large pleural effusion, and in no case was aspiration necessary or attempted. In most cases the fluid had a plastic appearance and presumably was a fibrinous exudate.

An increase in hilar density or accentuation of the pulmonary vascular shadows was noted in only 11 of the cases. This is in sharp contrast to atypical pneumonia, where it is probably the earliest and most frequent finding (9, 10). Elevation and distortion of the hilus occurred only once, and in this case there was some reason to believe that there may have been pre-existing path-

Fig. 4. G. M., age 54, Caucasian male; gardener, living in dairy area. Sudden onset 9/15/47. Severe malaise, headache, moderate myalgia, slight weakness and drowsiness, fever and chills. Sore throat at onset. Slight cough. No pleural pain. Admitted to hospital 9/18/47. Acutely ill; prostrated. Positive Brudzinski and Kernig; moderately stiff neck. Bronchial breathing right mid-thorax. White blood cells 7,540 (polymorphonuclears 70%, lymphocytes 28%, monocytes 2%). Maximum fever 102.6° F. Duration of fever nine days. Q fever complement-fixation titer: 9/19/47 positive 1:8; 10/6/47 positive 1:128.

Roentgenogram made on fifth day of illness, 9/19/47. Mottled consolidation of the medial basal segment of the right lower lobe. Accentuation of the minor interlobar fissure.

ological changes. Elevation of the diaphragm on the involved side occurred in 5 cases. None of the cases showed evidence of either hilar or mediastinal lymphadenopathy.

Most of the patients were admitted to the various hospitals a considerable period of time after the onset of their illness. From the information which is available, it is apparent that the pneumonic consolidations appear early in the course of the disease. The one patient examined a day after the onset of clinical symptoms had a consolidation of the mediobasal segment of the left upper lobe. Three patients were examined on the second day after the onset. The first showed a segmental consolidation, the second a solitary patch of pneumonia, and the third had a normal chest On re-examination of the latter patient, after an interval of several days, there was still no evidence of pneumonia. No patients were examined on the first day of their illness.

While, as noted above, there was little or no tendency for the lesions to be migratory, the area of involvement frequently increased by direct extension. Though this could occur as long as the patient remained febrile, it was more likely to do so early rather than late in the disease.

Resolution began in most cases with the return of the temperature to normal.

Fig. 5. A. P., age 23, Negro male; autopsy assistant. No history of exposure. Sudden onset 10/27/47. Moderate malaise, slight anorexia, vomiting, fever and chills. Very severe headache and slight drowsiness. No cough, pleural pain, or upper respiratory symptoms. Admitted to hospital 10/30/47. Acutely ill. Moderately stiff neck and positive Brudzinski. No pulmonary findings on physical examination. White blood cells 12,550 (polymorphonuclears 73%, lymphocytes 26%, monocytes 1%). Erythrocyte sedimentation rate 42 mm./hr. Maximum fever 103.2° F. Duration of fever eight days. Q fever complement-fixation titer: 11/4/47 negative; 11/13/47 positive 1:32; 12/16/47 positive 1:256.

Roentgenogram made on tenth day of illness. Homogeneous consolidation of right upper lobe.

Seven days after the cessation of fever, partial resolution had occurred. At the end of two months there were residual strand shadows. Six months later these were completely absorbed.

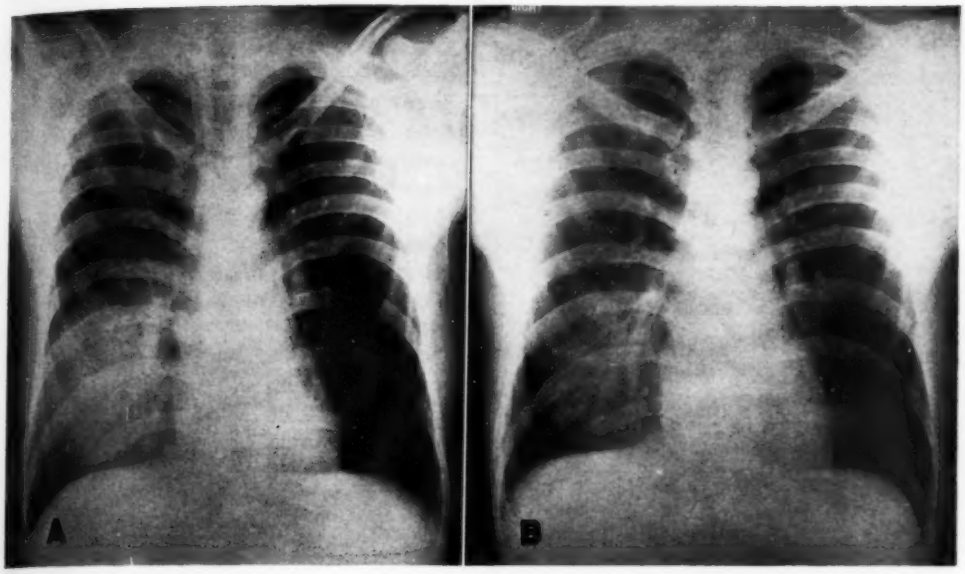

Fig. 6. F. L., age 29, Caucasian male; mechanic. No history of exposure. Sudden onset 4/12/47. Moderate malaise, anorexia, headache, slight cough, chills and fever. Pleural pain in right chest. No upper respiratory symptoms. Admitted to hospital 4/17/47. Acutely ill, listless, and confused. Dullness and râles at right base. White blood cells 4,400 (polymorphonuclears 87%, lymphocytes 10%, monocytes 3%). Erythrocyte sedimentation rate 38 mm./hr. Maximum fever 105° F. Duration of fever twelve days. Q fever complement-fixation titer: 6/19/47 positive 1:128.

A. Roentgenogram made on sixth day of illness, 4/17/47. Segmental homogeneous consolidation of right lower lobe, with accentuation of the minor interlobar fissure. Three days after the cessation of fever slight resolution had occurred.

B. Roentgenogram made 7/21/47, three months after cessation of fever. Residual linear infiltration.

Varying degrees of resolution were evident often within the first few days after the cessation of fever or occasionally even preceded it. In a case in which the fever lasted for eleven days, a roentgenogram of the chest taken on the tenth day of illness showed almost complete resolution. In one with fever for fifteen days, there was partial resolution on the thirteenth day.

It is difficult to determine the time required for complete resolution from the data available, inasmuch as only 4 cases were recorded as being clear at the time of the last roentgenogram. Thirty-five of the 65 patients with pneumonia had more than one roentgenogram of the chest. Six of these showed evidence of delayed resolution varying from twenty to sixty days after the return of the temperature to normal. Five were noted as having persistent changes, consisting of linear strand shadows.

CORRELATION OF CLINICAL AND ROENTGEN FINDINGS

The clinical severity of each case was graded from 1 plus to 4 plus, based on duration of fever, degree of prostration, presence of confusion and delirium, and the general impression of the clinician attending the patient.

Roentgenographic extent of involvement was based on the amount of lung involved: 0, no evidence of pneumonia; 1 plus, a single pulmonary segment or less; 2 plus, more than one pulmonary segment but less than one lobe (except right middle lobe); 3 plus, one lobe (except right middle lobe); 4 plus, more than one lobe.

There was some tendency for those with the greater extent of pneumonic infiltration to be the more ill (Table I). A similar distribution of cases was seen when comparing roentgenographic extent and the leukocyte count (Table II), and the dura-

TABLE I: SHOWING TENDENCY FOR THOSE WITH THE GREATER EXTENT OF PNEUMONIC INVOLVEMENT TO BE THE MORE ILL

Clinical Severity	Roentgenographic Extent of Involvement* 0	+	++	+++	++++
+	5	7	7	..	..
++	4	8	13	4	..
+++	2	7	6	3	1
++++	1	2	3	1	3

* 0 Negative. + Single pulmonary segment or less. ++ More than one segment but less than one lobe (except middle lobe). +++ One lobe (except middle lobe). ++++ More than one lobe.

tion of fever (Table III). There was no correlation between bilateral involvement and clinical severity.

Chest pain was frequent and was a complaint of 30 patients. In 12 it was associated with pleural fluid. Eighteen patients with chest pain had no visible pleural fluid and 8 with pleural fluid did not complain of chest pain. Where recorded, the location of the pain corresponded to that of pneumonia. Three patients who did not have pneumonia had chest pain. In 2 of these it was substernal.

TABLE II: SHOWING TENDENCY FOR THOSE WITH THE GREATER EXTENT OF PNEUMONIC INVOLVEMENT TO HAVE HIGHER WHITE BLOOD COUNTS

White Blood Count	Roentgenographic Extent of Involvement* 0	+	++	+++	++++
4000– 4999	2	4	2	..	..
5000– 5999	4	4	3	1	..
6000– 6999	..	3	3	..	..
7000– 7999	2	6	3	..	..
8000– 8999	1	2	3	2	1
9000– 9999	..	1	4	..	1
10000–10999	..	2	3	2	1
11000–11999	..	..	2	..	..
12000–12999	..	1	2	1	..
13000–13999	1	1	..	..	1
14000–14999	1	..	3	2	..
15000–15999	1	..	..	..	..
16000–16999	..	..	..	..	..
17000–17999	..	..	1	..	..

* See footnote to Table I.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Q fever cannot be differentiated by means of its symptomatology or clinical and routine laboratory findings from many other acute febrile illnesses. It is most often confused with primary atypical and virus pneumonias, influenza, and meningitis. Because of the minimal respiratory symptoms, many cases of Q fever with pneumonia are overlooked, particularly those presenting the symptom complex of severe headache and fever with or without associated meningism. The diagnosis should be suspected in any acute febrile episode the etiology of which is not readily established by bacteriologic, serologic, or other means. This is true whether or not

TABLE III: SHOWING TENDENCY FOR THOSE WITH THE GREATER EXTENT OF PNEUMONIC INVOLVEMENT TO HAVE A GREATER DURATION OF FEVER

Duration of Fever (days)	Roentgenographic Extent of Involvement 0	+	++	+++	++++
4	..	..	..	1	..
5	1	..	..	..	..
6	1	..	2	..	..
7	3	3	2	..	..
8	3	4	2	..	..
9	..	4	5	..	..
10	..	2	3	2	..
11	..	5	3	2	..
12	..	3	2	2	1
13	1	1	1	1	..
14	1	2	3	..	1
15	..	..	4	..	..
16	..	..	..	..	1
17	1	..	1	..	..
18	..	..	..	..	..
19	..	..	..	..	..
20	..	..	..	..	1
21–23	..	..	..	..	..
24–26	..	..	..	..	..
27–30	1	..	..	..	..

there has been contact with the animal industry or unpasteurized milk.

Roentgenologically it is not possible, in the majority of cases, to distinguish pneumococcic pneumonia from that of Q fever. Both usually present segmental or lobar consolidations of varying density, accentuated towards the periphery of the lung fields. An associated fibrinous pleuritis is common to both. The pneumonia of Q fever tends to resolve more slowly than that due to the pneumococcus.

Although clinically atypical pneumonia and Q fever present a somewhat similar picture, roentgenologically they are quite different. The hilar and vascular engorgement so frequently seen in atypical pneumonia is notably absent in Q fever.

Segmental or lobar infiltrations may be present in either disease but are more common in Q fever. Homogeneous consolidations are the most frequent lesions in Q fever as contrasted to the more patchy and mottled infiltrations of atypical pneumonia. These are frequently migratory in atypical pneumonia but are seldom so in Q fever. In either disease, multiple lobes may be involved and resolution may be prolonged.

Some of the more severe fungous diseases, including coccidioidomycosis and blastomycosis, primary tuberculosis, and histoplasmosis, usually show, in addition to their pulmonary parenchymal lesions, hilar or mediastinal lymphadenopathy. This is absent in Q fever. Neither cavitation nor pneumatocele formation was observed in any of our cases. With resolution, both the fungous diseases and tuberculosis may leave residua of rounded discrete foci, linear strand shadows, or calcifications both in lung parenchyma and regional lymph nodes. Only residual linear strand shadows have thus far been observed in Q fever. Sputum studies, skin and serologic tests serve further to differentiate these diseases.

A definite diagnosis of Q fever is possible only by recovery of the causative organism, *Coxiella burnetii*, from the blood stream, or by demonstration on successive examinations of the blood of a rising Q fever complement-fixation titer. An initial negative reaction in the first week of illness does not exclude the disease. A single positive reaction of a titer of 1:32 or higher during the convalescent phase of a clinically compatible illness is strong presumptive evidence of Q fever.

Fig. 7. B. G., age 39, Caucasian male; owner of a fat-rendering plant. Diagnosis of Hodgkin's paragranuloma established six months prior to present illness. Sudden onset 5/18/48. Marked weakness, severe headache, chest pain, and fever. No upper respiratory symptoms. Admitted to hospital 5/25/48. Acutely ill, cyanotic. Dullness and râles at right base. White blood cells 8,000 (polymorphonuclears 63%, lymphocytes 37%). Maximum fever 104° F. Duration of fever fourteen days. *Coxiella burnetii* recovered from blood stream, 6/1/48. Q fever complement-fixation titer: 5/29/48 negative; 6/1/48 positive 1:8; 6/2/48 positive 1:64.

Roentgenogram made on ninth day of illness, 5/26/48. Cloudy homogeneous consolidation of right lower lobe; small area of consolidation in left lower lobe; mottled cloudy consolidation in medial portion of left upper lobe. Small amount of fibrinous exudate at right base, extending into minor interlobar fissure.

Two days after the cessation of fever the exudate at the right base had increased. Resolution was gradual but was not complete until two months later.

SUMMARY

The clinical features of Q fever are discussed briefly. It cannot be differentiated clinically from many other acute febrile illnesses and is most often confused with primary atypical and virus pneumonias, influenza, and meningitis.

A study of the roentgenograms of the chests of 77 cases of Q fever admitted to various hospitals in Southern California is presented. Sixty-five patients (84 per cent) had pneumonia of varying extent. Thirteen (24 per cent) of those with pneumonia had bilateral involvement. Spread of the pneumonia by direct extension occurred frequently. Migratory lesions were rarely encountered. There was a tendency for those with the greater extent of pneumonic involvement to be the more ill, have higher white blood counts, and a greater duration of fever.

The most characteristic radiographic lesion (83 per cent) was a homogeneous segmental or lobar consolidation. An increase in hilar density or accentuation of the pulmonary vascular shadows was no-

tably absent. Minor amounts of predominantly fibrinous pleural exudate were seen in almost one-third of the cases. None showed evidence of either hilar or mediastinal lymphadenopathy.

Pneumonic consolidations have appeared as early as one day after the onset of clinical symptoms.

While resolution usually began with the return of the temperature to normal and might be completed within a short period of time, it was not infrequently delayed.

Roentgenologically, Q fever closely resembles pneumococcic pneumonia. It often differs considerably in appearance from the atypical pneumonias.

A definite diagnosis of Q fever can be made only on isolation of the Rickettsia (*Coxiella burnetii*) from the blood stream of the patient or on demonstration of specific antibodies during convalescence.

NOTE: We wish to acknowledge gratefully the generosity of the following radiologists for the privilege of using their cases, which gave us a much more adequate sampling of the roentgen manifestations of Q fever than could have been obtained from our material alone: Joseph Jellen, M.D., St. Francis Hospital, Lynwood, and Downey Memorial Hospital, Downey; Lowell S. Goin, M.D., Queen of the Angels Hospital, Los Angeles; Charles W. McClanahan, M.D., Wadsworth General Hospital, Veterans Administration, Los Angeles; Clayton R. Johnson, M.D., Murphy Memorial Hospital, Whittier; Hubert J. Prichard, M.D., Seaside Memorial Hospital, Long Beach.

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BIBLIOGRAPHY

1. BECK, M. D., BELL, J. A., SHAW, E., AND HUEBNER, R. J.: Q Fever Studies in Southern California. II. An Epidemiologic Study of 300 Cases. To be published.

2. BROWN, D. C., KNIGHT, L. A., AND JELLISON, W. L.: A Fatal Case of Q Fever in Southern California. California Med. **69:** 200–202, September 1948.

3. BURNET, F. M., AND FREEMAN, M.: Experimental Studies on the Virus of Q Fever. M. J. Australia **2:** 299–305, Aug. 21, 1937.

4. CHENEY, G., AND GEIB, W. A.: Identification of Q Fever in Panama. Am. J. Hyg. **44:** 158–172, July 1946.

5. COMMISSION ON ACUTE RESPIRATORY DISEASES: A Laboratory Outbreak of Q Fever Caused by the Balkan Grippe Strain of Rickettsia burneti. Am. J. Hyg. **44:** 123–157, July 1946.

6. DAVIS, G. E., AND COX, H. R.: Filter-Passing Infectious Agent Isolated from Ticks. Isolation from Dermacentor andersoni, Reactions in Animals and Filtration Experiments. Pub. Health Rep. **53:** 2259–2267, Dec. 30, 1938.

7. DENLINGER, R. B.: Clinical Aspects of Q Fever in Southern California. Ann. Int. Med. **30:** 510–527, March 1949.

8. DERRICK, E. H.: "Q" Fever, New Disease Entity: Clinical Features, Diagnosis and Laboratory Investigation. M. J. Australia **2:** 281–299, Aug. 21, 1937.

9. DINGLE, J. H., ET AL.: Primary Atypical Pneumonia, Etiology Unknown. Am. J. Hyg. **39:** 67–125, January 1944; 197–258, March 1944; 269–331, May 1944.

10. FINLAND, M., AND DINGLE, J. H.: Medical Progress: Virus Pneumonia. I. Pneumonias Associated with Known Non-Bacterial Agents: Influenza, Psittacosis and Q Fever. New England J. Med. **227:** 342–350, Aug. 27, 1942.

11. DYER, R. E., TOPPING, N. H., AND BENGTSON, I. A.: Institutional Outbreak of Pneumonitis. Isolation and Identification of Causative Organism. Pub. Health Rep. **55:** 1945–1954, Oct. 25, 1940.

12. FEINSTEIN, M., YESNER, R., AND MARKS, J. L.: Epidemics of Q Fever among Troops Returning from Italy in the Spring of 1945. I. Clinical Aspects of the Epidemic at Camp Patrick Henry, Virginia. Am. J. Hyg. **44:** 72–87, July 1946.

13. GSELL, O.: Pneumonias a Rickettsia burneti. Méd. et hyg. **108:** 317, October 1947.

14. HORNIBROOK, J. W., AND NELSON, K. R.: Institutional Outbreak of Pneumonitis. Epidemiological and Clinical Studies. Pub. Health Rep. **55:** 1936–1944, Oct. 25, 1940.

15. HUEBNER, R. J., ET AL.: Q Fever Studies in Southern California. I. Recovery of Rickettsia burneti from Raw Milk. Pub. Health Rep. **63:** 214–222, Feb. 13, 1948.

16. HUEBNER, R. J.: Unpublished data.

17. IRONS, J. V., AND HOOPER, J. M.: Q Fever in the United States: Clinical Data on an Outbreak Among Stock Handlers and Slaughterhouse Workers. J. A. M. A. **133:** 815–818, March 22, 1947.

18. LENNETTE, E. H.: Q Fever in California. California Med. **69:** 91–95, August 1948.

19. LENNETTE, E. H., AND MEIKLEJOHN, G.: Q Fever in Central and Northern California. California Med. **69:** 197–199, September 1948.

20. LILLIE, R. D., PERRIN, T. L., AND ARMSTRONG, C.: An Institutional Outbreak of Pneumonitis. Histopathology in Man and Rhesus Monkeys in the Pneumonitis Due to the Virus of "Q" Fever. Pub. Health Rep. **56:** 149–155, Jan. 24, 1941.

21. LILLIE, R. D.: Pathologic Histology in Guinea Pigs Following Intraperitoneal Inoculation with the Virus of "Q" Fever. Pub. Health Rep. **57:** 296–300, Feb. 27, 1942.

22. ROBBINS, F. C., AND RAGAN, C. A.: Q Fever in the Mediterranean Area. Report of Its Occurrence in Allied Troops. I. Clinical Features of the Disease. Am. J. Hyg. **44:** 6–22, July 1946.

23. SHEPARD, C. C.: Outbreak of Q Fever in a Chicago Packing House. Am. J. Hyg. **46:** 185–192, September 1947.

24. YOUNG, F. W.: Q Fever in Artesia. Los Angeles County Health Index, May 17, 1947. Also in California Med. **69:** 89, August 1948.

SUMARIO

Manifestaciones Roentgenológicas de la Fiebre (Rickettsiasis) Q

En una breve reseña de las características clínicas de la fiebre Q, señálase que no puede diferenciarse el mal clínicamente de otras muchas afecciones febriles agudas, confundiéndose más a menudo con neumonías virales y atípicas primarias, influenza y meningitis.

Preséntase también un estudio de las radiografías torácicas de 77 enfermos con fiebre Q, admitidos en varios hospitales del sur de California. Trece (24 por ciento) de los que tenían neumonía mostraron invasión bilateral. La propagación de la neumonía por difusión directa fué frecuente. Rara vez se observaron lesiones migratorias. En los que era más extensa la invasión neumónica, notóse tendencia a mayor gravedad, más altas fórmulas leucocitarias y mayor duración de la fiebre.

La más típica lesión radiográfica (83 por ciento) consistió en hepatización segmentaria o lobular homogénea. Brillaron por su ausencia el aumento de las condensaciones hiliares y la acentuación de las imágenes de los vasos pulmonares. Casi en la tercera parte de los casos observáronse pequeñas cantidades de exudado pleural de predominio fibrinoso. En ninguno había signos de linfadenopatía hiliar o mediastínica. Las hepatizaciones pulmonares aparecieron hasta al día de la iniciación de los sintomas.

Aunque la resolución solió comenzar con la normalización de la temperatura y fué a veces completa en breve período de tiempo, también se demoró con bastante frecuencia.

Roentgenológicamente, la fiebre Q se asemeja a la neumonía neumocócica, discrepando muy a menudo considerablemente en su apariencia de las neumonías atípicas.

Un diagnóstico preciso de fiebre Q sólo puede hacerse al aislar la rickettsia (Coxiella burnetii) de la sangre del enfermo o encontrar los anticuerpos específicos durante la convalecencia.

EDITORIAL

Palliative Roentgen Therapy

It is generally unwise to attempt to do the impossible when malignant disease is far advanced, but the decision to institute radiation therapy should bear with it the responsibility of doing the job thoroughly or not at all.

Since the majority of patients suffering from malignant disease come under the care of the radiation therapist at some time or other, it is important for him to try to obtain the greatest possible degree of palliation of symptoms, even if there is no chance for the eradication of the malignant process. The statement has been made that approximately 70 per cent of patients with deep-seated malignant disease coming to hospitals today are unsuitable for curative surgery.

The decision to use radiation therapy does not depend entirely upon the criteria of inoperability. All patients with inoperable tumors are not necessarily suitable for palliative radiation therapy, but the fact remains that most cases are in that category. If the radiation therapist uses his best skill and judgment in the application of suitable and adequate irradiation, the malignant process will occasionally be arrested, perhaps eradicated; many patients will receive a satisfactory degree of palliation, but the largest number will steadily decline and die of the disease. This is probably why the lay-public has such a universal fear of cancer, in spite of all the radio, newspaper, and magazine propaganda. Through continued education and the overcoming of fear by the activities of the various cancer societies and related organizations, it can be hoped that the two more favorable groups will constantly enlarge as a result of earlier diagnosis and institution of energetic treatment, especially where the disease is localized.

A planned program of irradiation includes a multitude of factors, among which the technical aspects of administration of the radiation are the particular responsibility of the radiation therapist. For one reason or another, usually laudable, his colleagues may try to persuade him to give radiation in unsuitable cases. Just how far he should go in the treatment of a patient whose condition is almost certainly hopeless must be decided in each individual case. Probably too many of these unfortunate persons are accepted because little if anything else can be done, but if they are accepted, the treatments should be carried to their full tolerable limits when this is necessary. The series should not be limited merely to avoid sharp radiation reactions which, after all, are of only temporary nature. Too often this is done. Because for a minor reason someone wishes to discontinue a course which has just started, the very purpose for which radiation was being given may be defeated. Alterations in the plan may be necessary as treatments continue, and the radiation therapist should be ready to make changes at any time to allow for transfusions, tapping, or adjunctive chemotherapy or supportive treatment to fit the particular situation.

Close medical management requires the cooperation of the attending or referring physician, who should be kept informed so that treatment of side-effects or complications and concurrent disease unrelated to the primary neoplasm can be dealt with promptly. The care of the radiation reactions after the course is completed, in those cases requiring more vigorous local irradiation, is the responsibility of the radiation therapist, and his follow-up examinations in these cases may be necessary to differentiate between recurrence

of the neoplasm and changes incident to irradiation.

The more one studies the human biologic effects of radiation, the more the fact stands out that the greater the total *quantity* administered, *i.e.*, the "tumor dose," the greater is the probability of a satisfactory response, so far as some malignant neoplasms are concerned. A striking impression is gained that the degree of palliation is much greater when irradiation follows surgery as closely as possible and when the treatment is carried to the full clinical extent of tolerance. Rather than "token therapy," with its limited physiologic and probably psychologic effects, a full course of treatment should be given.

Instances are occasionally seen in which palliation of pain is not obtained, though it should have resulted, as in the local irradiation of osseous metastases from carcinoma of the breast or of osseous involvement by the lymphomatoid diseases. The total exposure given was insufficient; if it had been greater, a more satisfactory palliation probably would have resulted. For some particular reason, these patients needed more irradiation than usual. If a lesion does not respond to the customary moderate exposure, there is no reason why more irradiation, even double the amount usually needed, should not be given until a satisfactory regression is started or the skin tolerance is reached. The fallacy of thinking that if a given "dose" produces a certain percentage of good results, an increased exposure should give increasingly better results, should, however, be avoided. This is not necessarily true. Eventually the optimum doses for various tumors will be learned; there is probably a maximum beyond which no benefit is obtained.

If the patient's general condition is bad, it is best to forego irradiation until improvement can be obtained by blood transfusions or other supportive measures, or the amount given at first should be small and gradually increased to a large total exposure, even over a period of two or three months if necessary. It is not good treatment to produce a prolonged annoying wet desquamation of the skin in an unfavorable case, but this can be avoided and controlled by proper timing of the exposures and the use of the smallest field which will cover the disease adequately.

For many malignant neoplasms the radiologist might say, "The larger the 'tumor dose,' the greater the probable degree of palliation."

HAROLD W. JACOX, M.D.

EASTERN CONFERENCE OF RADIOLOGISTS

The next meeting of the Eastern Conference of Radiologists will be held in Boston on Friday and Saturday, March 3 and 4, 1950, as voted by the delegates of the participating societies at the Washington meeting. The local committee is preparing an excellent program. Complete details will appear at an early date.

WISCONSIN RADIOLOGICAL SOCIETY

A new society of radiologists of the state of Wisconsin was organized at a meeting held in Madison, on Aug. 26, 1949. The organization is called the Wisconsin Radiological Society and has thirty-five charter members, all of whom are diplomates of the Board of Radiology. The following officers were elected: Dr. Lawrence V. Littig of Madison, President; Dr. Ernst A. Pohle of Madison, President Elect; Dr. Irving I. Cowan of Milwaukee, Secretary-Treasurer; Board of Censors, Dr. S. Richard Beatty of Neenah, Dr. J. Edwin Habbe of Milwaukee, and Dr. Victor J. Bruder of La Crosse.

SOCIEDAD BRASILEIRA DE RADIOTERAPIA

The Brazilian Society of Radiotherapy has recently announced the election of its new officers for 1949–50: Dr. Andrelino Amaral, President; Dr. Oscar R. von Pfuhl, Secretary; Dr. Renato Araujo Cintra, Treasurer. Meetings are held on the second Wednesday of the month at 9:00 P.M., at the Society's headquarters, Av. Brigadeiro Luis Antonio, 644, São Paulo.

OAK RIDGE INSTITUTE OF NUCLEAR STUDIES

The Special Training Division of the Oak Ridge Institute of Nuclear Studies announces three additional basic courses in the technic of using radioisotopes as tracers, continuing a series offered during the past year and a half. The courses will run from Jan. 2 to 27, Jan. 30 to Feb. 25, and March 6 to 31, 1950. Thirty-two participants will be accepted for each course. Application for the first course (Jan. 2 to 27) should be mailed prior to Dec. 1.

Each session is divided into laboratory work, lectures on laboratory experiments, general background lectures, and special-topic seminars. Ample time is allowed for library work and conferences with the staff on individual problems. Seminar topics include the use of tracers in animal and human experimentation, design of radiochemical laboratories, dosimetry, instrumentation, the effects of radiation on living cells, and the principles and practices of radiation protection.

A registration fee of $25.00 is charged for each participant. Hotel or dormitory facilities are available in Oak Ridge at standard rates.

Application forms and additional information on the courses may be obtained from Dr. Ralph T. Overman, Chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tenn.

GEORGE EASTMAN HOUSE

The George Eastman House, Rochester, New York, a public educational institute to further the knowledge of photography's means, accomplishments, and potentialities in all fields, was formally opened to the public on Nov. 9. The house itself is a beautiful Georgian Colonial structure originally built as a residence of the late George Eastman. In addition to housing important collections of photographs, photographic equipment, apparatus for research, and historically important literature on photography, the Institute offers facilities for exhibitions, demonstrations, lectures, and the showing of motion pictures, conferences on photography, and research in its history, science, and application.

In the Hall of Contemporary Photography, which adjoins the main building, are special exhibits illustrative of such subjects as nuclear physics, astronomy, x-ray and electron diffraction, spectrography, photomicrography and medical photography, radiography, and photofluorography.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

PHYSICS AND THE SURGEON. By H. S. SOUTTAR, D.M., M.Ch. (Oxon.), F.R.C.S. (Eng.), Hon. M.D., Trinity College, Dublin; Hon. F.R.A.C.S., Consulting Surgeon, London Hospital. Publication No. 48, American Lecture Series, edited by Michael E. DeBakey, M. D., and R. Glen Spurling, M.D. A volume of 60 pages, with 41 illustrations. Published by Charles C Thomas, Springfield, Ill., 1948.

ATOMIC MEDICINE. Edited by CHARLES F. BEHRENS, M.D., Captain, MC, U. S. Navy; Director, Atomic Defense Division, Bureau of Medicine and Surgery, Navy Department; Medical Officer in Command, Naval Medical Research Institute, National Medical Center, Bethesda, Md. A volume of 416 pages, with numerous illustrations. Published by Thomas Nelson & Sons, New York, 1949. Price $7.50.

TOMOGRAFIA CLINICA CARDIO-VASCULAR. By DR. JUAN GOVEA, Cardiologist, Hospital Mercedes, Havana, and DR. FIDEL AGUIRRE, Radiologist, Institute of Radium, Hospital Mercedes, Havana. Library of Medical Practice Vol. 29. A volume of 486 pages, with 308 illustrations. Published by Cultural, S.A., 525 Obispo St., Havana, Cuba, 1949. Price $8.00.

ANNUAL REPORT ON THE RESULTS OF RADIOTHERAPY IN CANCER OF THE UTERINE CERVIX. FIFTH VOLUME. STATEMENTS OF RESULTS OBTAINED IN 1941 AND PREVIOUS YEARS (collated in 1948). Sponsored by The British Empire Cancer Campaign, London; The Donner Foundation, Philadelphia; The Cancerföreningen, Stockholm; and The World Health Organization. Editorial Committee: DR. J. HEYMAN (Editor) Stockholm; DR. M. DONALDSON, London; DR. L. C. SCHEFFEY, Philadelphia. A volume of 245 pages. Published by P. A. Norstedt & Söner, Stockholm, 1949.

DIE PATHOLOGIE DES HARNLEITERS IM RÖNTGENBILD. By DR. ANTON THELEN. A monograph of 88 pages, with 71 illustrations. Published by Georg Thieme, Stuttgart, 1949. Distributed by Grune & Stratton, Inc., New York. Price $3.25.

ÜBER DIE NEUEN STRAHLENSCHUTZREGELN FÜR DIE HERSTELLUNG UND ERRICHTUNG MEDIZINISCHER RÖNTGENEINRICHTUNGEN UND -ANLAGEN, DIN 6811 UND 6812. By HERBERT GRAF, Chief Engineer of the Siemens-Reiniger Works, Erlangen. A monograph of 72 pages. Published by Georg Thieme, Stuttgart, 1949. Distributed by Grune & Stratton, Inc., New York. Price $1.25.

EXPERIMENTELLE UNTERSUCHUNGEN ÜBER RÖNTGENEFFEKTE UND CHEMISCHE EFFEKTE AUF DIE PFLANZLICHE MITOSE. By DR. KURT HOHL. A monograph of 88 pages, with 30 illustrations and tables. Published by Georg Thieme, Stuttgart, 1949.

UNTERSUCHUNGEN ÜBER DEN LUMBALEN UND CERVIKALEN WIRBELBANDSCHEIBENVORFALL. By DR. F. FREISCHAUER, Chief of the Surgical Clinic of the City Hospital, Essen. A monograph of 88 pages, with 25 illustrations. Published by Georg Thieme, Stuttgart, 1949. Distributed by Grune & Stratton, Inc., New York. Price $3.00.

Book Reviews

RADIOACTIVE TRACER TECHNIQUES. By GEO. K. SCHWEITZER, Associate Professor of Chemistry, University of Tennessee, and Oak Ridge Institute of Nuclear Studies, and IRA B. WHITNEY, Chief Supervisor of Radio Chemistry Process Department, Oak Ridge National Laboratories. A volume of 242 pages, with 13 figures. Published by D. Van Nostrand Company, Inc., Toronto, New York, and London, 1949. Price $3.25.

This book is designed as a guide for laboratory work and instruction in the utilization of radioactive tracers. It is planned for use by a class directed and supervised by a competent instructor, and it is assumed that the laboratory course will be accompanied by a suitable lecture course. It should be valuable for such groups, but it also contains much that will be useful to the laboratory worker who must of necessity acquire certain radioactive technics without formal help.

The first four chapters are devoted to basic considerations regarding radiation hazards and the design, construction, and operation of a radiolaboratory. Then follow detailed directions for four groups of experiments: (*a*) basic manipulations with electroscopes and counters; (*b*) chemical experiments, including exchange reactions, analysis by isotopic dilution, and chemical effects of radiation; (*c*) physical experiments, including half-life and absorption measurements; (*d*) biological experiments, including radioautographs and uptake and excretion studies. Instructions are concise and clear; the line diagrams of the apparatus are simple and helpful. Each experiment is preceded by a brief account of the theory involved and followed by a series of questions and a few pertinent references. Appendices contain lists of apparatus, lists of sources of supply, and convenient multiplication tables.

Some criticism should be directed toward the extreme precautionary measures recommended in the first section. It is true that the need for safe handling procedures must be drilled into every individual concerned in any way with radioactive isotopes. The question, in any particular instance, is what constitutes safety. It is stated in the first chapter that, "since this book is primarily for the beginner, only the tracer level laboratories will be considered in detail," yet some of the procedures given are appropriate only in radiochemical laboratories handling considerable amounts of active material. This bias has appeared regularly in articles on safe handling procedures coming from the large atomic energy centers, and it is understandable that it should be so. Their rules were formulated under stress of emergency conditions, little was known of possible consequences, stringent regulations could be enforced, it was felt desirable to make them, and they worked. It appears that as yet too little thought has been given to possible modifications of these to meet needs for smaller and simpler laboratories. It is the contention of this reviewer that any safety regulations should be set up to be reasonable *for the case in hand*. Such regulations will be observed, while unreasonably stringent ones will be by-passed.

Cases in point are the requirements that *anyone entering the laboratory* must wear personal monitoring meters, and that suitable protective clothing should be provided for all *visitors* (italics the reviewer's). The change of clothing for students entering this type of laboratory is not practicable, nor necessary. On the other hand, it is essential to keep a close check on contamination of persons, clothing, or books, as much for the sake of keeping experiments clean, and for inculcating good habits, as for actual safety. For the student taking such a course, there seems to be little reason for monthly blood counts; the wearing of monitor instruments will warn sooner of possible danger. Every effort should be made to give the student a wholesome respect for the dangers inherent in carelessness with radiations, at any level, but inculcation of unreasoning fear should be avoided.

This book should prove valuable for every teacher including a few or many radiation experiments in a laboratory course, for anyone training technicians in radioactive procedures, and for all those scientists who are being obliged to train themselves in the use of these substances.

DIE INDIKATIONEN ZUR RÖNTGEN UND RADIUM-BESTRAHLUNG. By DR. MED. HABIL. R. GLAUNER, Dozent für Röntgenologie und Strahlenheilkunde, Stuttgart. A volume of 128 pages. Published by Georg Thieme, Stuttgart, 1948. In paper cover DM 7.20.

In the introduction to this monograph on roentgen and radium irradiation, the author states that after almost half a century of radiation therapy the time has come when a critical review of its present status seems to be in order. While he addresses his remarks also to radiologists, his main purpose is to provide physicians in general practice and in other specialties with definite indications and contraindications for the use of roentgen rays and radium in the treatment of malignant and non-malignant disease. Therefore, all physical and technical details have been omitted, thus facilitating the perusal of the text by the non-radiologist. Some of the thoughts expressed by the author in the introduction are certainly worth mentioning. He defends the right and the duty of the radiologist to refuse cases unsuitable for irradiation and points out that the mere availability of the necessary apparatus must not influence such a decision. He also emphasizes the fact, which is still overlooked at times both here and abroad, that therapeutic radiology should be practised only by those properly trained and qualified.

The subject matter has been arranged under the following headings: Indications in Malignant Disease; in Localized Inflammations, Tuberculosis and Actinomycosis; and Indications in Other Diseases. These last include diseases of the blood, skin, glands of internal secretion, bones and joints, gynecological disorders, diseases of the male sex organs, the nervous system, the circulatory system, the lungs, the gastro-intestinal tract, and the eyes. The use of roentgen rays in gas gangrene and diphtheria carriers is discussed briefly. The "therapeutic test irradiation" in the differential diagnosis of mediastinal tumors is also mentioned. In three tables are shown the five-year survivals in the most frequently seen malignant diseases, their incidence in males and females, and recommendations for the therapeutic approach, be it surgery, surgery plus irradiation, or irradiation alone.

The American radiologist will be inclined to agree with a good many of the views expressed by the author, but there are occasions where opinions are apt to differ. While it is not possible to go into detail, a few examples may be mentioned.

In most clinics, carcinoma of the cervix, stage I, is operated on; others use a combination of x-rays and radium. Carcinoma of the fundus is considered a surgical subject and irradiation is given following operation; a few clinics favor preoperative irradiation also. In carcinoma of the lip and tongue the cosmetic results of radiation therapy are considered better than with surgery. The author strongly favors preoperative irradiation in seminoma of the testicle. He is very pessimistic regarding the prognosis in osteogenic sarcoma and feels that simple resection plus intensive irradiation is as good or better than amputation. In the discussion of inflammatory diseases he stresses the value of irradiation of the tonsils, especially in people with frequent attacks of tonsillitis. Therapy should be administered in the symptom-free interval. In some forms of tuberculosis, *e.g.*, of the cervical nodes and female pelvis, good results have been reported; however, the former enthusiasm in Germany for irradiation of pulmonary tuberculosis has disappeared.

Among the contraindications for radiation therapy the author lists acne, sycosis barbae, hyperhidrosis, psoriasis, and lupus erythematosus.

This monograph should prove of interest to all who desire to become acquainted with the present status of radiation therapy in Germany. Throughout the text a strictly conservative attitude is maintained, and the strongly subjective presentation does not detract from its value. After all, in it is reflected the author's credo as a radiotherapist who has the earnest desire to do what is best for his patients. A brief bibliography is appended.

RADIOLOGICAL SOCIETIES: SECRETARIES AND MEETING DATES

Editor's Note: Secretaries of state and local radiological societies are requested to co-operate in keeping this section up-to-date by notifying the editor promptly of changes in officers and meeting dates.

RADIOLOGICAL SOCIETY OF NORTH AMERICA. *Secretary-Treasurer*, Donald S. Childs, M.D., 713 E. Genesee St., Syracuse 2, N. Y.

AMERICAN RADIUM SOCIETY. *Secretary*, Hugh F. Hare, M.D., 605 Commonwealth Ave., Boston 15, Mass.

AMERICAN ROENTGEN RAY SOCIETY. *Secretary*, Harold Dabney Kerr, M.D., Iowa City, Iowa.

AMERICAN COLLEGE OF RADIOLOGY. *Secretary*, William C. Stronach, 20 N. Wacker Dr., Chicago 6, Ill.

SECTION ON RADIOLOGY, A. M. A. *Secretary*, U. V. Portmann, M.D., Cleveland Clinic, Cleveland 6, Ohio.

Alabama

ALABAMA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. D. Anderson, M D , 420 10th St., Tuscaloosa.

Arizona

ARIZONA ASSOCIATION OF PATHOLOGISTS AND RADIOLOGISTS. *Secretary*, R. Lee Foster, M. D., 507 Professional Bldg., Phoenix.

Arkansas

ARKANSAS RADIOLOGICAL SOCIETY. *Secretary*, Fred Hames, M.D., Pine Bluff. Meets every three months and at meeting of State Medical Society.

California

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY. *Secretary*, Sydney F. Thomas, M.D., Palo Alto Clinic, Palo Alto.

EAST BAY ROENTGEN SOCIETY. *Secretary*, Dan Tucker, 434 30th St., Oakland 9. Meets monthly, first Thursday, at Peralta Hospital.

LOS ANGELES RADIOLOGICAL SOCIETY. *Secretary*, Wybren Hiemstra, 1414 S. Hope St. Meets monthly, second Wednesday, County Society Bldg.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB. *Secretary*, Robert L. Ayers, M.D., 726 4th St., Marysville. Meets at dinner last Monday of September, November, January, March, and May.

PACIFIC ROENTGEN SOCIETY. *Secretary*, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually with State Medical Association.

SAN DIEGO ROENTGEN SOCIETY. *Secretary*, R. F. Niehaus, M.D., 1831 Fourth Ave., San Diego. Meets first Wednesday of each month.

X-RAY STUDY CLUB OF SAN FRANCISCO. *Secretary*, Wm. F. Reynolds, M.D., University Hospital, San Francisco 22. Meets third Thursday at 7:45, January to June at Stanford University Hospital, July to December at San Francisco Hospital.

Colorado

COLORADO RADIOLOGICAL SOCIETY. *Secretary*, Paul E. RePass, M.D., 306 Republic Bldg., Denver 2. Meets monthly, third Friday, at University of Colorado Medical Center or Denver Athletic Club.

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, Fred Zaff, M.D., 135 Whitney Ave., New Haven. Meetings bimonthly, second Wednesday.

CONNECTICUT VALLEY RADIOLOGICAL SOCIETY. *Secretary*, Ellwood W. Godfrey, M.D., 1676 Boulevard, W. Hartford. Meets second Friday of October and April.

District of Columbia

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY. *Secretary*, Karl C. Corley, M.D., 1835 Eye St., N.W., Washington 6. Meets third Thursday, January, March, May, and October, at 8:00 P.M., in Medical Society Auditorium.

Florida

FLORIDA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John J. McGuire, M.D., 1117 N. Palafox, Pensacola. Meets in April and in November.

Georgia

ATLANTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Wm. W. Bryan, M.D., 490 Peachtree St., N. E. Meets second Friday, September to May.

GEORGIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Robert Drane, M.D., De Renne Apartments, Savannah. Meets in November and at the annual meeting of State Medical Association.

Illinois

CHICAGO ROENTGEN SOCIETY. *Secretary*, John H. Gilmore, M. D., 720 N. Michigan Ave., Chicago 11. Meets at the University Club, second Thursday of October, November, January, February, March, and April at 8:00 P.M.

ILLINOIS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, William DeHollander, M.D., St. Johns' Hospital Springfield. Meetings quarterly as announced.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, Harold L. Shinall, M.D., St. Joseph's Hospital, Bloomington.

Indiana

INDIANA ROENTGEN SOCIETY. *Secretary-Treasurer*, William M. Loehr, M.D., 712 Hume-Mansur Bldg., Indianapolis 4. Annual meeting in May.

Iowa

IOWA X-RAY CLUB. *Secretary*, Arthur W. Erskine, M.D., 326 Higley Building, Cedar Rapids. Meets during annual session of State Medical Society.

Kansas

KANSAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Anthony F. Rossitto, M.D., Wichita Hospital, Wichita. Meets annually with State Medical Society.

Kentucky

KENTUCKY RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Everett L. Pirkey, M.D., 323 East Chestnut St., Louisville 2.

LOUISVILLE RADIOLOGICAL SOCIETY, *Secretary-Treasurer*, Everett L. Pirkey, Louisville General Hospital, Louisville 2. Meets second Friday of each month at Louisville General Hospital.

Louisiana

LOUISIANA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Johnson R. Anderson, M.D., No. Louisiana Sanitarium, Shreveport. Meets with State Medical Society.

ORLEANS PARISH RADIOLOGICAL SOCIETY. *Secretary*, Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets first Tuesday of each month.

SHREVEPORT RADIOLOGICAL CLUB. *Secretary*, Oscar O. Jones, M.D., 2622 Greenwood Road. Meets monthly September to May, third Wednesday.

Maryland

BALTIMORE CITY MEDICAL SOCIETY, RADIOLOGICAL SECTION. *Secretary*, J. Howard Franz, M.D., 1127 St. Paul St., Baltimore 2.

Michigan

DETROIT X-RAY AND RADIUM SOCIETY. *Secretary-Treasurer*, George Belanger, M.D., Harper Hospital, Detroit 1. Meetings first Thursday, October to May, at Wayne County Medical Society club rooms.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS. *Secretary-Treasurer*, R. B. MacDuff, M.D., 220 Genesee Bank Building, Flint 3.

Minnesota

MINNESOTA RADIOLOGICAL SOCIETY. *Secretary*, C. N. Borman, M.D., 802 Medical Arts Bldg., Minneapolis 2. Meets in Spring and Fall.

Missouri

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY. *Secretary*, Wm. M. Kitchen, M.D., 1010 Rialto Building, Kansas City 6, Mo. Meetings last Friday of each month.

ST. LOUIS SOCIETY OF RADIOLOGISTS. *Secretary*, Charles J. Nolan, M.D., 737 University Club Bldg. Meets on fourth Wednesday, October to May.

Nebraska

NEBRASKA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Ralph C. Moore, M.D., Nebraska Methodist Hospital, Omaha 3. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England

NEW ENGLAND ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, George Levene, M.D., Massachusetts Memorial Hospitals, Boston. Meets monthly on third Friday at the Harvard Club.

New Hampshire

NEW HAMPSHIRE ROENTGEN SOCIETY. *Secretary*, Albert C. Johnston, M.D., Elliot Community Hospital, Keene. Meetings quarterly in Concord.

New Jersey

RADIOLOGICAL SOCIETY OF NEW JERSEY. *Secretary*, Benjamin Copleman, M.D., 280 Hobart St., Perth Amboy. Meets at Atlantic City at time of State Medical Society and midwinter in Elizabeth.

New York

ASSOCIATED RADIOLOGISTS OF NEW YORK, INC. *Secretary*, William J. Francis, M.D., East Rockaway.

BROOKLYN ROENTGEN RAY SOCIETY. *Secretary*, J. Daversa, M.D., 603 Fourth Ave., Brooklyn. Meets fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meetings second Monday, October to May.

CENTRAL NEW YORK ROENTGEN SOCIETY. *Secretary*, Dwight V. Needham, M.D., 608 E. Genesee St., Syracuse 10. Meetings January, May, October.

KINGS COUNTY RADIOLOGICAL SOCIETY. *Secretary*, Marcus Wiener, M.D., 1430 48th St., Brooklyn 19. Meetings fourth Thursday evening, October to May, at 8:45 P.M., in Kings County Medical Bldg.

NEW YORK ROENTGEN SOCIETY. *Secretary*, F. H. Ghiselin, M.D., 111 E. 76 St., New York.

QUEENS ROENTGEN RAY SOCIETY. *Secretary*, Jacob E. Goldstein, M.D., 88-29 163rd St., Jamaica 3. Meets fourth Monday of each month.

ROCHESTER ROENTGEN-RAY SOCIETY. *Secretary-Treasurer*, Ralph E. Alexander, M.D., 101 Medical Arts Bldg., Rochester 7. Meets at Strong Memorial Hospital, third Monday, September through May.

North Carolina

RADIOLOGICAL SOCIETY OF NORTH CAROLINA. *Secretary*, James E. Hemphill, M.D., Professional Bldg., Charlotte 2. Meets in May and October.

North Dakota

NORTH DAKOTA RADIOLOGICAL SOCIETY. *Secretary*, Charles Heilman, M.D., 1338 Second St., N., Fargo.

Ohio

OHIO STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Edward C. Elsey, M.D., 927 Carew Tower, Cincinnati 2. Meets with State Medical Association.

CENTRAL OHIO RADIOLOGICAL SOCIETY. *Secretary*, Paul D. Meyer, M.D., Grant Hospital, Columbus. Meets second Thursday, October, December, February, April, and June, 6:30 P.M., Seneca Hotel, Columbus.

CINCINNATI RADIOLOGICAL SOCIETY. *Secretary*, Eugene L. Saenger, M.D., 735 Doctors Bldg., Cincinnati 2. Meets last Monday, September to May.

CLEVELAND RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John R. Hannan, M.D., Cleveland Clinic, Cleveland 6. Meetings at 6:30 P.M. on fourth Monday, October to April, inclusive.

Oklahoma

OKLAHOMA STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. E. Brown, M.D., 21st and Xanthus, Tulsa 4. Meets in October, January, and May.

Oregon

OREGON RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Boyd Isenhart, M.D., 214 Medical-Dental Bldg., Portland 5. Meets monthly, on the second Wednesday, at 8:00 P.M., in the library of the University of Oregon Medical School.

Pacific Northwest

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Sydney J. Hawley, M.D., 1320 Madison St., Seattle 4. Meets annually in May.

Pennsylvania

PENNSYLVANIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, James M. Converse, M.D., 416 Pine St., Williamsport 8. Meets annually.

PHILADELPHIA ROENTGEN RAY SOCIETY. *Secretary*, George P. Keefer, M.D., 1930 Chestnut St., Philadelphia 9. Meets first Thursday of each month at 8:00 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY. *Secretary-Treasurer*, R. P. Meader, M.D., 4002 Jenkins Arcade, Pittsburgh 22. Meets second Wednesday of each month at 6:30 P.M., October to June.

Rocky Mountain States

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Maurice D. Frazer, M.D., Lincoln Clinic, Lincoln, Nebr.

South Carolina

SOUTH CAROLINA X-RAY SOCIETY. *Secretary-Treasurer*, Robert B. Taft, M.D., 103 Rutledge Ave., Charleston 16.

South Dakota

RADIOLOGICAL SOCIETY OF SOUTH DAKOTA. *Secretary-Treasurer* Marianne Wallis, M.D., 1200 E. Fifth Ave., Mitchell. Meets during Annual Session of State Medical Society.

Tennessee

MEMPHIS ROENTGEN CLUB. Meetings second Tuesday of each month at University Center.

TENNESSEE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, J. Marsh Frére, M.D., 707 Walnut St., Chattanooga. Meets annually with State Medical Society in April.

Texas

DALLAS-FORT WORTH ROENTGEN STUDY CLUB. *Secretary*, X. R. Hyde, M.D., Medical Arts Bldg., Fort Worth 2. Meets monthly, third Monday, in Dallas odd months, Fort Worth even months.

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Utah

UTAH STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Angus K. Wilson, M.D., 343 S. Main St., Salt Lake City. Meets third Wednesday, January, March, May, September, November.

Virginia

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Washington

WASHINGTON STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John H. Walker, M.D., 1115 Terry Ave., Seattle. Meetings fourth Monday, October through May, at College Club, Seattle.

Wisconsin

MILWAUKEE ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, Theodore J. Pfeffer, M.D., 839 N. Marshall St., Milwaukee 2. Meets monthly on second Monday at the University Club.

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UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE. Meets first and third Thursdays 4 P.M., September to May, Service Memorial Institute, Madison 6.

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SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA. Offices in Hospital Mercedes, Havana. Meets monthly.

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SOCIEDAD MEXICANA DE RADIOLOGÍA Y FISIOTERAPIA. *General Secretary*, Dr. Dionisio Pérez Conio, Marsella 11, México, D. F. Meetings first Monday of each month.

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ROENTGEN DIAGNOSIS

THE HEAD AND NECK

Porencephaly in Institutional Epileptics. Albert W. Pigott, Thomas S. P. Fitch, and Samuel M. Weingrow. J. Nerv. & Ment. Dis. **108:** 496–501, December 1948.

Four hundred epileptics who showed jacksonian characteristics, auras, or neurologic signs suggestive of a focal lesion in their seizures, were subjected to encephalography in a search for porencephaly. This was found in 20 cases. These 20 cases were then studied in detail.

Clinically the patients showed hemiplegia and sensory changes, both present from birth. Although the left side was involved in the majority, few showed any speech defect. Plain films of the skull showed diminished capacity of the cranium on the involved side. The paranasal sinuses were larger on that side, and the orbital and petrous ridges were higher, as well as the lesser wing of the sphenoid. The feature which differentiates porencephaly from the other conditions in which the findings so far enumerated are also found (unilateral brain atrophy, unilateral ventricular dilatation, and relapsing juvenile hematoma) is the thinness of the cranial bones on the involved side. They are thickened in the other conditions mentioned.

On encephalography the air-filled cavity in the brain substance is visualized, communicating with the lateral ventricle. None of the findings discussed are illustrated.

ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Fibro-Osteomas of the Paranasal Sinuses, Roentgenologically Simulating Malignant Neoplasms. Sölve Welin. Acta radiol. **30:** 457–463, Dec. 31, 1948.

The author presents three cases of histologically proved fibro-osteoma of the paranasal sinuses. In one, the process was in the ethmoid region, and in two the maxillary sinus was involved. In all cases, there was distinct osseous destruction due to pronounced pressure atrophy.

It is important that fibro-osteoma be considered in the differential diagnosis of lesions which produce bone destruction in the paranasal sinuses, because of their resemblance to malignant neoplasms.

Five roentgenograms. P. B. LOCKHART, M.D.
Indiana University

Roentgen Diagnosis of Otitis Media and Mastoiditis in Infancy and Early Childhood. Barton R. Young. Pennsylvania M. J. **52:** 213–214, December 1948.

Mastoid cells are rarely demonstrable before the sixth month of life and there are usually too few cells present to make a positive diagnosis of mastoiditis until after the tenth month. The diagnosis of otitis media, on the other hand, can be substantiated in the early months of life, because the tympanic cavity is relatively large and the petrous bone is not sufficiently ossified to obscure changes due to the disease. After the eighteenth month, the mastoid begins to resemble the adult type and the tip becomes pneumatized. Infection will cause a retardation or complete arrest of cellular development.

A diagnosis of mastoiditis cannot be made by roentgen evidence alone, because opacity of the mastoid can be produced by conditions other than inflammatory changes. It is relatively easy to demonstrate cloudiness in a pneumatic type of mastoid when the other side is taken for comparison, but when both sides are involved, the diagnosis is more difficult.

Early bone absorption is difficult to see, but advanced destruction is easily detected.

Evaluation of a chronically diseased mastoid is difficult, particularly if it is underdeveloped, as the sclerotic bone obscures the few cells that are present.

Roentgen examination is indicated when petrositis, cholesteatoma, otogenic meningitis, and brain abscess are suspected, as opacities or destruction of the mastoid or petrous pyramid are frequently demonstrable.

JOSEPH T. DANZER, M.D.
Oil City, Penna.

Localization of Foreign Bodies in the Eye by Bone-Free Radiography. K. Lindblom. Acta ophth. **26:** 439–440, 1948. **Roentgenographic and Fluoroscopic Control of the Extraction of Foreign Bodies from the Eye During Operation.** K. Lindblom. Ibid., pp. 441–443.

These two brief papers review technics of examination previously reported. The first describes a modification of the Vogt method of bone-free radiography of the eye in which a mentovertical projection is combined with the usual lateral view (Lindblom: Acta radiol. **15:** 615, 1934). The second describes the method of Larsson (Acta ophth. **19:** 1, 1941), in which two tangential roentgenograms are obtained during operation, and the fluoroscopic examination that is sometimes required to supplement this procedure.

One illustration accompanies each article.

Clinical and Roentgenological Views Regarding the Localization of Foreign Bodies in the Orbit. H. Larsson, K. Lindblom, and S. Stenström. Acta ophth. **26:** 557–558, 1948. **A. Variations in the Size and Shape of the Eye.** Sölve Stenström. Ibid., pp. 559–567. **B. Bi-Tangential Method of Roentgenographic Localization of Foreign Bodies in the Eye (Vertico-mental and Lateral Projections).** K. Lindblom. Ibid., pp. 568–573. **C. Localization of Foreign Bodies in the Orbit with Concurrent Determination of the Size of the Bulb.** H. Larsson. Ibid., pp. 574–580.

After a general discussion of the roentgenologic localization of foreign bodies in the orbit, each author takes up one aspect of the subject.

A. Stenström describes measurements of a large number of eyes of varying refraction, including both the axial length (according to Rushton: Trans. Ophth. Soc. United Kingdom **58:** 136, 1948) and the horizontal diameter (according to the method of Berg: Arch. f. Ophth. **127:** 606, 1931). The variation of these two quantities is analyzed, and also their correlation. It is concluded that the size and form of the eye show such marked variation that it is impossible in all cases to reckon with a bulb of average size in localizing orbital foreign bodies. Not even if the refraction of the eye is known can one work with a schematic value of the axial length. Where the greatest precision is required, and especially in the borderline cases where the foreign body lies close to the posterior pole of the eye, one should, if possible, measure the length and equatorial diameter of the bulb. If this is impracticable, the variations in the size of the eye should at any rate be taken into due account. Four charts are included.

B. A new geometric method of localization of foreign bodies in the eye by bi-tangential projections is described by Lindblom. These projections are obtained by different positions of the tube. The head is fixed, and the direction of vision is constant, being kept at right angles to the direction of projection. A small primary diaphragm and film without intensifying screens are used. Outlines of normal eyeballs, corresponding in size to the degree of magnification, are engraved on a transparent film. The appropriate eyeball outline is placed on the roentgenogram and the position of the foreign body is marked on it. The distance of the latter, in millimeters, from the coronal, sagittal, and horizontal midplanes of the eyeball is then measured. The distance from the coronal midplane should be equal on all films, otherwise new ones must be taken. If the distance from the coronal midplane is a, from the sagittal midplane is b, and from the horizontal one is c, the distance from the center of the bulb is $\sqrt{a^2+b^2+c^2}$. A figure smaller than 12 indicates an intra-ocular position of the foreign body. The distances b and c on a coronal diagram show the position in the coronal plane. The distance a, from the coronal midplane, gives the distance from the corneal border.

Eight roentgenograms; 2 photographs.

C. Rushton's technic for measuring the length of the eyeball, based on the principle that roentgen rays stimulate the retina, has been modified by Larsson for use in patients with foreign bodies in or close to the eyeball. His method for localizing foreign bodies in the orbit, with concurrent determination of the size of the bulb, is described in detail.

Two photographs.

Dermoid and Epidermoid Tumors of the Orbit. Raymond L. Pfeiffer and Russell J. Nicholl. Arch. Ophth. **40:** 639–664, December 1948.

Dermoid and epidermoid tumors of the orbit are uncommon if paraorbital growths or those located superficially in the eyelids are excluded. In a series of 200 consecutive cases of exophthalmos reported by one of the authors (Am. J. Ophth. **26:** 724, 816, and 928, 1943) the incidence of these congenital growths was 4 per cent. When intraorbital, these tumors displace the eyeball, and the present paper is limited to a discussion of tumors of this type. Nine cases are presented. The differentiation of dermoids and epidermoids is discussed and an attempt is made to clarify the terminology. Because there is little embryologic and histologic distinction between these growths, it is contended that the term "dermoid" is sufficient to cover both. Intermediate or transitional types are common.

All 9 dermoids in the present series were cystic, with epithelial linings varying greatly in number of layers and appearance of cells, and with fluid and semisolid contents. Hair was found in only 1 case.

Dermoids or epidermoids may arise in the diploe of the skull or bones of the orbit and in their growth expand both the inner and the outer table and produce rather characteristic defects. They may arise subperiosteally or subperiorbitally and cause fossae or indentations of the bone with typical markings. These defects may be demonstrated roentgenographically and serve to indicate the nature of the process and, in case of involvement of the orbit, the cause of the resulting exophthalmos. The authors' series of 9 cases, all verified, illustrates the importance and nature of the bony involvement in the diagnosis and treatment of these growths. Five additional unmistakable cases have been studied, but have not yet been verified histologically.

In their most typical form, the defects in the bone of the orbit produced by these tumors are revealed roentgenographically by sharply demarcated margins, with increase in the density of the bone at the border and diminished tissue density within the confines of the lesion itself. When there is associated dehiscence of bone, and, therefore, lack of calcium, the dark appearance of the growth on the film is striking.

The frontal bone was involved in 7 of the 9 cases; in 6, the growth appeared to have arisen in the diploe of the bone forming the roof of the orbit. In 2 cases a through-and-through defect in the bone was produced, so that pulsation of the dura was observed at operation. An hourglass tumor was found in 1 case, with portions both in the frontal fossa and in the orbit. The largest tumor appeared to have arisen in the diploe of the roof and to have grown mainly upward, to occupy most of the frontal fossa of that side. In several cases the adjacent frontal sinus was encroached upon, but in each instance the sinus was clear and the wall of the growth could be well distinguished.

In the differential diagnosis, several conditions must be considered. Retention cyst or mucocele of the frontal sinus may be distinguished by its connection with the sinus and the clouded condition of the sinus itself. A xanthoma of the bone or an eosinophilic granuloma does not satisfy the criteria for dermoids, especially in respect to the increase in the density of the bone at the margins of the lesion. Invasion by a malignant tumor, meningocele, and saccular aneurysm of the internal carotid artery must also be kept in mind.

Since dermoids of the orbit vary in size, position, and composition, and may involve neighboring structures and cavities, their treatment requires thorough study of the case and careful planning. In 6 of the 9 cases, direct approach through incision at the margin of the orbit over the presenting mass was adequate. The lesion was incised and evacuated, the remaining sac was phenolized, next treated with alcohol, and irrigated with saline solution. The cyst wall was then removed as completely as possible. Because of the toxic character of the contents of the cyst, aspiration not only will fail to improve matters but may produce intractable inflammation in the orbit.

Twelve roentgenograms; 9 drawings; 7 photographs; 3 photomicrographs; 1 table.

THE CHEST

Diagnostic Fluoroscopy at a Chest Clinic. W. H. Tattersall. Lancet **2:** 974–975, Dec. 18, 1948.

The author weighed the objections to the use of fluoroscopy for the diagnosis of pulmonary tuberculosis —(1) risk of x-ray injury to the observer; (2) lack of a permanent record; (3) the perceptive limitations of the human eye in dim light—against the advantages and decided to offer the general practitioners in a town of 100,000 a rapid fluoroscopic service. Physicians referring patients for examination were promised a report by return mail.

During the first two years of operation of the clinic, 2,908 patients were screened. Of this number, 90 per cent were discharged forthwith. Patients found to have non-pulmonary abnormalities were discharged and their further investigation left to their physicians. No patient who was passed as normal has subsequently

been found to have tuberculosis. Altogether 314 patients were selected for further study, including appropriate x-ray films. Of these, 83 (26 per cent) were found to have pulmonary tuberculosis, an incidence of 3 per cent of the total number sent for fluoroscopy; in only 32 of these was the sputum test positive. Thus a substantial proportion of relatively early cases have been discovered in this way.

Mass X-ray Examination Using Miniature Films. Clifton Hall. Illinois M. J. **94:** 369–372, December 1948.

The author presents some figures as to the work of the Division of Tuberculosis Control of the Illinois Department of Public Health. In mobile x-ray surveys in the sixteen months beginning with January 1946, more than 170,000 examinations were made. Among the points brought out are the following:

1. Films of 35 mm., 70 mm., and 4 × 5 in. are equally acceptable for screening purposes.
2. Stereoscopic miniatures are not necessary; the single film is entirely adequate.
3. The survey film detects but is not diagnostic.
4. Tuberculosis is not a disease found commonly in children.
5. The prevalence of tuberculosis increases with age, particularly after the age of twenty.
6. Age specific death rates increase in the older groups.
7. Surveys should be confined to those above fifteen years of age, and the number of children be kept at a minimum.
8. The survey process should be concentrated among industrial and other general adult population groups.
9. The mass survey must not replace the standard x-ray examination or the medical training and experience of the practitioner.

EDWARD E. LEVINE, M.D.
Dearborn, Mich.

Studies of Pulmonary Findings and Antigen Sensitivity Among Student Nurses. III. Pulmonary Infiltrates and Mediastinal Adenopathy Observed Among Student Nurses at the Beginning of Training. Lydia B. Edwards, Ira Lewis, and Carroll E. Palmer. Pub. Health Rep. **63:** 1569–1600, Dec. 3, 1948.

This report is based on an analysis of chest radiographs and tuberculin and histoplasmin sensitivity tests on 12,803 student nurses in ten of the larger cities of the country. It is admitted that this is a highly selected group in which the incidence of positive findings would be expected to be under that of the general population because of the medical "screening" of applicants at the time of entrance into the training period.

A reactor to tuberculin was defined as one having 5 or more mm. of definite induration to a 0.0001 mg. dose of P.P.D-S (purified protein derivative), and a reactor to histoplasmin as one showing any erythema or palpable induration to a similar amount of 1/1,000 histoplasmin.

The pulmonary infiltrates shown radiographically were classified into four broad groups: (*a*) nodular; (*b*) poorly circumscribed; (*c*) fibrotic; (*d*) "non-specific." The last group included obvious pneumonia, bronchiectasis, etc., clearly unrelated to tuberculosis and histoplasmin infection.

In 224 of the entire number examined, definite pulmonary infiltrates were demonstrable on the original radiographic studies. The percentage rate for the very large part of the entire group which showed no reaction to either antigen was very low, 0.26 per cent, while that of the positive tuberculin reactors was 3.51 per cent, that of histoplasmin reactors was 4.98 per cent, and that of those reacting to both antigens was 4.68 per cent.

The incidence of reactors to histoplasmin varied widely in different localities from as low as 6.3 per cent to as high as 53.4 per cent, while the incidence for tuberculin reactors varied from 4.5 per cent to 14.6 per cent.

In studying the relationship of nodular infiltrates to antigen reactions, it was found that the percentage rate was 3.44 per cent in reactors to histoplasmin alone, as compared with 0.41 per cent in reactors to tuberculin alone, and 0.05 per cent in non-reactors.

The rates for poorly circumscribed infiltrates were 1.88 per cent among reactors to tuberculin alone, 0.44 per cent among reactors to histoplasmin alone, and 0.03 per cent among non-reactors to both antigens.

For fibrotic infiltrates the rates were 1.22 per cent among reactors to tuberculin alone, 0.95 per cent among reactors to histoplasmin alone, and 0.07 per cent among non-reactors. As would be expected, there was no significant relation between sensitivity and the type of infiltrate in the "non-specific" group.

There was a definite tendency for infiltrates associated with tuberculin sensitivity to be localized to the upper parts of the lung fields, particularly those of the nodular type.

Thirty-eight of the entire group showed mediastinal adenopathy. Thirty-one of these reacted to histoplasmin alone, 4 to both histoplasmin and tuberculin, 3 to neither, and none to tuberculin alone. Seventeen had the so-called "primary complex"—mediastinal adenopathy associated with parenchymal infiltrate. Of these, 14 were reactors to histoplasmin alone, 3 reacted to both histoplasmin and tuberculin, and again none to tuberculin alone.

Twenty-three of the entire group had infiltrates of the poorly circumscribed type, associated with positive tuberculin reaction. Of these, 7 showed definite increase in the size of the infiltrate during periods of observation ranging from one to three years.

This is an important article, particularly for those doing chest survey work, and should be read in its entirety.

Fourteen radiographs; 11 tables; 5 figures.

BERNARD S. KALAYJIAN, M.D.
Detroit, Mich.

Pathogenesis of Minimal Pulmonary Tuberculosis. A Study of 1,225 Necropsies in Cases of Sudden and Unexpected Death. E. M. Medlar. Am. Rev. Tubere. **58:** 583–611, December 1948.

This study details the results of examination of the lungs from 1,225 necropsies of individuals over twenty years of age dying suddenly and unexpectedly, with reference to the pathogenesis of minimal tuberculous lesions. Only lesions which were large enough so that they might have been expected to have caused detectable roentgenographic shadows were considered. Lesions were divided into primary and re-infection types. There were 105 persons with a single calcified complex and 96 with minimal disease, either primary or

re-infection. The results are presented in a series of tables and are summarized as follows:

1. Of the 96 persons with minimal lesions, the disease represented a primary infection in 42.7 per cent and a re-infection in 57.3 per cent. Of 23 persons less than forty years of age, the disease was primary in 91 per cent. Of 73 persons over forty years of age the disease was a re-infection in 72.6 per cent.

2. Evidence of tuberculous lesions in one or more abdominal viscera was found seven times more often in the primary than in the re-infection group, suggesting that extrapulmonary tuberculosis may be expected more often in persons with primary than with re-infection disease.

3. The gross appearances are similar for both primary and re-infection lesions, indicating that it is not possible from roentgenographic shadows to differentiate between the two.

4. In the group analyzed, only 13.8 per cent represented completely healed disease. Shadows caused by tuberculous disease should not be interpreted as "healed" if there are scattered small calcific densities, for the majority of such lesions also contain caseous areas of tuberculous pneumonia.

Nine roentgenograms; 10 photomicrographs; 1 graph; 5 tables.

L. W. PAUL, M.D.
University of Wisconsin

Tuberculosis Among Philadelphia Foodhandlers. Katharine R. Boucot and Martin J. Sokoloff. Am. Rev. Tuberc. **58:** 684–692, December 1948.

This article details the results of a survey for tuberculosis among the food handlers of Philadelphia. Approximately 32,000 persons were examined during a ten-month period; 70-mm. photofluorograms were obtained on all individuals and interpreted by a radiologist. The prevalence of re-infection tuberculosis was 2.3 per cent, being slightly higher in the whites than in the non-whites. Complete follow-up studies of all persons whose photofluorograms were interpreted as indicating tuberculosis or suspected tuberculosis confirmed the impression of tuberculosis in 76 per cent. The remainder were found to have non-tuberculous disease or no abnormalities whatsoever, or are still diagnostic problems.

Two roentgenograms; 3 charts; 2 tables.

L. W. PAUL, M.D.
University of Wisconsin

Coccidioidomycosis. Persistence of Residual Pulmonary Lesions. H. E. Bass, A. Schomer, and R. Berke. Arch. Int. Med. **82:** 519–528, December 1948.

The primary purpose of this article is to present data on a series of 20 patients residing in New York City with protracted residual lung lesions of coccidioidomycosis and to discuss the pitfalls in the diagnosis. The patients were veterans of World War II who had returned to civilian life. In the majority of instances the diagnosis of pulmonary coccidioidomycosis had been made prior to discharge from military service. Thirteen of the more representative cases are described.

Several pertinent observations were made in this study:

1. The residual pulmonary lesions were of all types, *i.e.*, nodular densities, cavities, mottled infiltrations, fibrosis, pleural effusion, and calcification.

2. These residual lesions characteristically showed little or no change over a period of observation of from two to five years.

3. All patients gave a history of residence in an endemic area, varying from three months to two years.

4. Skin sensitivity to coccidioidin diminished with the lapse of time.

5. A negative reaction to the coccidioidin skin test was misleading in the presence of a disseminating lesion. In such an instance, the failure to react to coccidioidin was presumed to be related to anergy. Serologic examination is usually diagnostic, however, and can often be confirmed by recovery of the spherules in a tissue biopsy, from sputum, or from draining sinuses.

6. In cases of cavity formation, no evidence of bronchogenic spread or of seeding to other parts of the lung fields, such as occurs commonly in tuberculosis, was observed.

7. The resemblance of the residual pulmonary lesions to those in tuberculosis was striking. In several of the cases, a diagnosis of tuberculosis had been made after the patient's return to civilian life, usually after a routine chest survey or pre-employment roentgenoscopic examination. In a number of instances, a positive reaction to both the tuberculin and the coccidioidin skin test made diagnosis difficult. In such cases a presumptive diagnosis of coccidioidomycosis was made on the following evidence: (*a*) history of exposure in an endemic area; (*b*) roentgen evidence of a pulmonary lesion, usually a solitary cavity or nodular density, which showed no change after months of observation; (*c*) indication of an identical lesion in the roentgenogram taken on separation from military service; (*d*) a positive reaction to the coccidioidin skin test; (*e*) absence of tubercle bacilli.

8. Almost all the patients in the series were asymptomatic. The notable exceptions included a patient with a cavity who had recurrent hemoptyses, a patient with pleural effusion, and the single patient with disseminated disease.

Six roentgenograms; 1 photograph.

Segmental Distribution of Bronchiectasis. H. E. Counihan. Irish J. M. Sc., pp. 731–737, November 1948.

The distribution of bronchiectasis, as revealed by adequate bronchograms, was studied in 149 cases. The lower lobes were, as usual, the most frequently involved. Right middle lobe and left lingular bronchiectasis were often associated with the lower lobe ectasia. The point is made that frequently the dorsal segments of the lower lobes are uninvolved (in 46 per cent of cases on the left side, 56 per cent on the right) and should not be resected. The author also mentions the occasional occurrence of ectasia of the pectoral segment of the upper lobe, requiring resection.

Four roentgenograms; 8 tables.

PAUL W. ROMAN, M.D.
Baltimore, Md.

Rheumatic Pneumonitis in Childhood. Harold B. Levy, John D. Coffey, and Charles E. Anderson, Jr. Pediatrics **2:** 688–693, December 1948.

Six cases of fatal rheumatic pneumonitis in children are presented, with autopsy findings in 5. In each of the 6 cases, it was felt that the clinical and pathologic findings justified the conclusion that the rheumatic pneumonitis was the major, if not the primary, cause of

death, either by impairment of the respiratory system alone or by adding further to the load of an already dangerously impaired cardiac mechanism.

During the course of rheumatic fever in childhood, especially in the initial attack, one must be constantly alert in an effort to discover the presence of widespread pulmonary involvement, as it would appear to have a grave prognostic significance.

The roentgen appearance of rheumatic pneumonitis is not appreciably different from pulmonary congestion, except for its transient character. The examination is chiefly of value in differentiating the condition from bacterial and primary atypical pneumonic consolidations.

Four photomicrographs; 1 table.

Problem of the Solitary Lung Tumors. Donald Brian Effler, Brian Blades, and Edward Marks. Surgery **24:** 917–928, December 1948.

Roentgen examination of the thorax in apparently normal individuals, as in the Armed Forces and in preventive medicine, has led to the diagnosis of increasing numbers of asymptomatic intrathoracic tumors. The authors present 24 cases of solitary asymptomatic lung neoplasms which were picked up on routine roentgen examinations of the chest, with subsequent operation.

Many doctors are reluctant to consider a patient with an asymptomatic lung tumor as a candidate for surgery, and the belief that solitary round masses in the lung fields are harmless is unfortunately widespread. However, in this series approximately 15 per cent of the tumors proved to be malignant, though all were considered benign preoperatively.

The question of proper diagnosis is difficult and can be answered only by tissue study of the entire lesion. With the continued improvement of surgical and anesthetic technic, exploratory thoracotomy has become an accepted procedure in all age groups. There seems to be little justification for overlooking the only certain method of diagnosis and treatment in the management of these tumors.

Eleven roentgenograms; 2 photographs; 1 table.

EDSEL S. REED, M.D.
Louisville, Ky.

Death Following Bronchography. Report of a Case. James A. Harrill and William B. Alsup. Ann. Otol., Rhin. & Laryng. **57:** 1088–1098, December 1948.

A case of death following the injection of iodized oil for bronchography is reported. The patient was a two-year-old boy whose chief complaints were cough, asthma, and frequent attacks of difficulty in breathing. Roentgenography showed a non-specific pneumonitis in the right base. Bronchoscopy revealed no abnormality beyond a mild hyperemia of the trachea and the main bronchi. Ten cubic centimeters of iodochloral were introduced through the bronchoscope without immediate incident other than the occurrence of cyanosis for forty seconds after about half the oil had been introduced. This subsided promptly, and the procedure was completed. The bronchial tree was well filled with oil except in the upper lobes. No evidence of bronchiectasis or abscess was obtained.

Thirty minutes after instillation of the oil, cyanosis developed rapidly, convulsive movements of all extremities occurred, respiration became rapid and labored, and death ensued in fifteen minutes, in spite of resuscitory measures. Tracheotomy was done, but the heart had stopped beating before the trachea was opened. It contained no secretion.

Autopsy revealed no free fluid nor adhesions in the pleural cavities. The heart contained no oil nor air. No thrombi were present in the pulmonary vessels. Both lungs were pink and homogeneous. No obstruction was found in the larger bronchi. Microscopic sections of the right lung showed considerable atelectasis. Within the bronchi and alveoli were large mononuclear cells containing red-staining fat. The trachea presented minimal edema. The final diagnosis was bilateral peribronchiolitis; interstitial pneumonitis; congestion of the kidneys; congestion of the spleen.

A review of the literature revealed other cases of reaction following instillation of iodized oil into the tracheobronchial tree, both fatal and non-fatal. Although the mechanism of death in many of the cases is not known, the presence of hypersensitivity to some constituent of the iodized oil is believed to exist, and the fatality here reported is thought possibly to have been due to this cause. There is no known test to pre-determine sensitivity to this medium.

It is concluded that bronchography should not be undertaken in an acutely ill patient or in the presence of demonstrable areas of pneumonitis. Precautions to be taken to guard against respiratory embarrassment in cases of sensitivity include the immediate availability of bronchoscopic means of removing the injected oil. Oxygen must be administered through the bronchoscope and under positive pressure. The stomach should be washed with salt solution if a roentgenogram reveals swallowed oil. Barbiturates may be administered intravenously for convulsions, adrenalin intramuscularly for its effect on the allergic reaction, and respiratory stimulants as indicated.

Two roentgenograms. STEPHEN N. TAGER, M.D.
Urbana, Ill.

An Angiographic Study of the Form and Function of the Remaining Lung After Pneumonectomy. Harold Neuhof and Robert A. Nabatoff. J. Thoracic Surg. **17:** 799–808, December 1948.

There is a difference of opinion concerning what happens to the remaining lung following pneumonectomy. Regeneration observed in young animals after pneumonectomy has as yet not been found in children. In adults some measure of hyperdistention of the lung and dilatation of the blood vessels is believed to occur, associated with mediastinal displacement. This, according to one group, interferes with function; according to another group, it does not. Almost all agree that a marked mediastinal shift causes compensatory changes in blood vessels and parenchyma and results in reduction in function, especially in older people. Consequently some advocate thoracoplasty to prevent or correct the mediastinal shift.

The authors studied 12 patients, chiefly in the older age group, who had undergone pneumonectomy for malignant tumors, but not thoracoplasty. The postoperative interval varied from six months to ten years. Angiograms were obtained according to Sussman's technic for angiocardiography. Six representative angiograms are reproduced in the article.

The authors conclude that there is no significant abnormality in size, shape, or distribution of the vascular pattern in the remaining lung after pneumonectomy. Moderate displacement of the mediastinum and great vessels occurred often in these cases, in which no

thoracoplasties were done. No clinical evidence of reduced pulmonary function was found. The expansion of the remaining lung, as judged by the vascular pattern, was not as great as has been described. No alteration in size or evidence of thrombosis in the stump of the resected pulmonary artery was observed.

HAROLD O. PETERSON, M.D.
University of Minnesota

Laminagraphy Following Thoracoplasty. Alessandro Piazza. Radiol. med. (Milan) **34:** 753–768, December 1948. (In Italian)

The author, having studied 50 cases in which thoracoplasty was done, using plain films and laminagraphic films, discusses the advantages and limitations of the laminagraphic method. Of special interest are the excellent laminagraphs of cross-sections of the chest obtained according to the method of Professor Vallebona. This method has been in clinical use for about two years, and the radiographs show transverse horizontal views at various levels of the thoracic cage.

Twenty-five illustrations.

CESARE GIANTURCO, M.D.
Urbana, Ill.

Gastric Cyst of the Mediastinum. Adrian A. Ehler and Susanna Atwell. J. Thoracic Surg. **17:** 809–820, December 1948.

The authors report one case of gastric cyst of the mediastinum and some statistics on the total cases reported to date, which number 27. Nineteen of the patients were infants under one year and only 2 were over fifteen years of age. This indicates that the lesion usually causes death early in life and hence should be removed.

The symptoms result largely from pressure on neighboring structures. Thus dyspnea, cough, and dysphagia are common complaints. Hemoptysis and hematemesis and melena may be present when the cyst communicates with the esophagus or trachea.

The roentgen examination is the most valuable diagnostic procedure. In 21 of the 27 cases, the mass projected into the right chest from the posterior superior mediastinum. In certain cases it may be advisable to aspirate the lesions preoperatively, as a diagnostic procedure. If hydrochloric acid is present, the nature of the lesion is established.

Total removal is considered the best treatment.

The authors' patient was a boy of 15 months. He had had difficulty eating and slight cough for two months and had passed several tarry stools. Massive hemorrhage of red blood from the mouth and rectum followed. The child preferred lying on the right side. Earlier roentgenograms showed a mass on the right side superiorly and medially, and subsequent films showed a fluid level in the mass. The tumor was aspirated and hydrochloric acid was found. The lesion was successfully removed. It measured 14 × 5.0 × 1.5 cm. and showed typical gastric mucosa grossly and microscopically. An ulcer 0.5 cm. in diameter was present in the upper end of the mass. Recovery was complete.

Three roentgenograms; 2 photographs; 2 photo micrographs.

HAROLD O. PETERSON, M.D.
University of Minnesota

Symposium on Anomalies of the Heart. Pulmonary Stenosis: Tetralogy of Fallot. Robert L. Parker. M. Clin. North America **32:** 855–877, July 1948. **Clinical-Pathologic Correlation of Some Less Common Cyanotic Congenital Cardiac Defects in Infants.** James W. DuShane. Ibid., pp. 879–894. **Atrial Septal Defects.** Thomas J. Dry. Ibid., pp. 895–910. **Variations in the Clinical and Pathologic Picture of Patent Ductus Arteriosus.** Howard B. Burchell. Ibid., pp. 911–923. **Anomalies of the Derivatives of the Aortic Arch System.** Jesse E. Edwards. Ibid., pp. 925–949.

Parker reports 4 cases of pulmonary stenosis. The first 2 cases, in children three years of age, illustrate the typical clinical features of pulmonary stenosis of the tetralogy of Fallot type, *i.e.*, cyanosis, secondary polycythemia, clubbing of the fingers and toes, and dyspnea on exertion, without enlargement of the heart. In the first child a successful result was obtained from a Blalock-Taussig anastomosis. In the second case, transient episodes of severe anoxemia with loss of consciousness were the prominent clinical feature. Pneumonia developed, and the child died shortly after hospital admission. Death was due primarily to anoxemia, the pneumonic process being a terminal phenomenon. Autopsy revealed the usual anatomic changes found in the tetralogy of Fallot, with the subpulmonic third ventricle clearly shown.

The third case, in a woman of twenty-four, with a minimal degree of incapacity, illustrates the adaptability of the circulation when the degree of stenosis is not severe. The fourth case, in a man of twenty-six, is of unusual interest not only because of the finding of pulmonary stenosis with intact ventricular septum but also because of a complicating brain abscess, to which death was due.

Seventeen illustrations, including 5 roentgenograms.

DuShane presents 4 cases, all of which came to necropsy, to demonstrate different types of uncommon congenital malformations of the heart. The first case was an atresia of the mitral valve with rudimentary left ventricle, patent foramen ovale, and ventricular septal defect; the second, aortic atresia with intact ventricular septum, patent foramen ovale, and patent ductus arteriosus; the third, atresia of the tricuspid orifice with rudimentary right ventricle, patent foramen ovale and ventricular defect; and the fourth, complete transposition of the aorta and pulmonary artery with patent ductus arteriosus and a defect in the membranous portion of the ventricular septum.

Clinical and pathological features of the cases are correlated in an effort to clarify the altered physiology and to emphasize the diagnostic features of mitral atresia, aortic atresia, tricuspid atresia, and transposition of the great vessels. The presence of cyanosis and the configuration of the heart on roentgenographic and roentgenoscopic examination provide the most accurate means of evaluation. Cardiac murmurs are of no importance in differentiating these anomalies in infancy, and the electrocardiogram is of little aid except in tricuspid atresia, in which the left axis deviation is diagnostic.

Four roentgenograms; 4 drawings; 5 electrocardiograms; 9 photographs of specimens.

Dry states that among congenital cardiac anomalies the incidence of atrial septal defects is rather high, even if one excludes cases in which the opening in the interatrial septum is merely large enough to permit the insertion of a probe and cases in which the opening is too small to produce secondary effects. A review of 133 cases of all types of congenital anomalies of the heart

and great vessels seen at the Mayo Clinic revealed 25 in which the atrial septal defect was large enough to produce symptoms. Eighteen of the 25 patients were females. Five cases of atrial septal defect are presented here, one with easily recognizable physical signs but with relatively mild symptoms; the second with incapacitation due to limited cardiac reserve and to many functional symptoms, with sudden and unexpected death; the third complicated by paroxysmal tachycardia with sudden death; the fourth complicated by cerebral abscess; and the fifth complicated by rheumatic mitral stenosis (Lutembacher's syndrome), auricular fibrillation, and congestive heart failure, with sudden death.

Three roentgenograms; 6 electrocardiograms; 2 photographs of specimens.

Burchell discusses the variations in the clinical and pathologic picture of patent ductus arteriosus and presents 2 illustrative cases. He draws attention to the irreversible changes that may occur in the pulmonary vascular tree or in a hypertrophied left ventricle, which may limit the completeness of the surgical cure if treatment has been long delayed.

Edwards describes and illustrates with drawings those anomalies of the derivatives of the aortic arch system which may interfere with the function of either the esophagus or the trachea. (Persistent patent ductus arteriosus and coarctation of the aorta are not included.) He divides malformations of the aortic arch derivatives into two main groups depending upon whether the ductus arteriosus (the term "ductus arteriosus" is used interchangeably with the term "ligamentum arteriosum") takes its origin from the left pulmonary artery or from the right pulmonary artery.

Sixteen drawings.

Combined Use of Angiocardiography and Cardiac Catheterization in the Diagnosis of Congenital Anomalies of the Cardiovascular System. Arnold L. Johnson and Donald L. McRae. Pediatrics **2:** 643–651, December 1948.

The findings on cardiac catheterization and angiocardiographic examination of 3 cyanotic children with congenital anomalies of the heart and great vessels are presented to demonstrate the value of these procedures, particularly in elucidating problems connected with the pulmonary artery. A fourth case, in which the diagnosis is not entirely clear, illustrates the relationship between the two methods. The patient was an eight-year-old Negro boy with a mediastinal mass and a history of heart murmur from birth and moderate reduction in exercise tolerance. This case shows evidence of pulmonary vein drainage into the superior vena cava. The question is raised as to whether the mass in the mediastinum represents an aneurysm of the superior vena cava or of a pulmonary vein, perhaps at the site of entry into the superior vena cava. No explanation can be offered for the left portion of the mass, which was not entered by dye or catheter.

Fifteen roentgenograms; 3 tables.

Familial Interauricular Septal Defect with Mitral Stenosis (Lutembacher's Syndrome). Sanford R. Courter, Benjamin Felson, and Johnson McGuire. Am. J. M. Sc. **216:** 501–508, November 1948.

Observations are reported on sisters, aged 21 and 26, with the typical physical and roentgenologic features of interauricular septal defect with mitral stenosis (Lutembacher's syndrome). No such familial occurrence has been previously reported.

Each of the sisters gave a history of chronic cardiac disease, rheumatic fever, and retarded development. Characteristic physical findings of bulging left precordium, increased pulmonary pressure (accentuated P2), systolic murmur over the pulmonic area, and the typical diastolic rumble of acquired mitral stenosis were found. Both patients were hypotensive. Roentgenologic findings were: (1) right-sided cardiac enlargement, (2) prominent pulmonary conus, (3) hilar prominence and "hilar dance," (4) small left ventricle, and (5) small to hypoplastic aorta.

Conspicuous in Lutembacher's syndrome is the absence of marked left auricular enlargement, since the defect acts as a safety valve in releasing the increased pressure within the left auricle resulting from the mitral valve stenosis.

A chest film, electrocardiogram, and phonocardiogram are reproduced for each case.

JAMES ALLAN READ, M.D.
The Henry Ford Hospital

Visualization of the Aorta and Its Branches by Retro-arterial Diodrast Injection. Thomas H. Burford and Merl J. Carson. J. Pediat. **33:** 675–687, December 1948.

A method of retro-arterial diodrast injection for the visualization of anomalies or disease of the aorta and its immediate branches is presented. Utilization of the left common carotid artery has proved satisfactory in 8 cases studied (all in children or young adults). This method provides a safe, dependable, and effective means of diagnosing questionable cases of patent ductus arteriosus, coarctation of the aorta, and aortic arch anomalies, and is useful in proving or disproving selected cases of aneurysm.

The technic includes general ether anesthesia, skin incision, dissection of the common carotid artery, placing of silk ligatures about the artery to prevent extravasation of blood and cephalad flow of the diodrast, rapid injection of 50 c.c. of 70 per cent diodrast through a cannula, roentgenographic exposures in the right posterior oblique position, and closure of the incisions of the artery and skin.

Arteriograms from six cases; 1 drawing.

ROBERT LARNER, M.D.
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THE DIGESTIVE SYSTEM

Radiographic Pneumoperitoneum in Acute Perforations of the Gastrointestinal Tract. Stanley A. Kornblum. New York State J. Med. **48:** 2726–2728, Dec. 15, 1948.

Some confusion exists in the literature in relation to the frequency of pneumoperitoneum after perforation of the gastro-intestinal tract. The author surveyed all the cases of gastro-intestinal perforation seen on the ward services at Mt. Sinai Hospital (New York) from 1933 to 1945. Unfortunately, among 322 cases, films were obtained in only 87.

The most frequent sites of perforation were, in order, the appendix, stomach, duodenum, large bowel, and jejunum and ileum. The percentage of cases showing free air were as follows: stomach, 59.3 per cent (27 x-ray examinations); duodenum, 70.4 per cent (27

x-ray examinations); large bowel, 63.6 per cent (11 x-ray examinations). Of 14 cases of appendiceal perforation for which films were obtained, only 1 showed pneumoperitoneum, and of 8 cases involving the jejunum and ileum, only 3 were positive in this respect. These figures are too small for significant conclusions.

The series was analyzed from the standpoint of the interval between onset and time of filming and the size of perforation. Neither of these factors played any significant part in determining whether pneumoperitoneum would be demonstrated.

It would be interesting if other hospitals with large numbers of cases would contribute their findings. This is a question which one is asked frequently, so it is well to have up-to-date figures.

Three tables. ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Gastroscopic Diagnosis of the Location and Extent of Gastric Cancer. C. Wilmer Wirts. Gastroenterology 11: 861–872, December 1948.

This is a study of 57 patients with gastric cancer, 40.2 per cent of whom showed either primary or secondary involvement of the cardia. All patients were subjected to roentgen as well as gastroscopic examinations. In 10 patients, or 17.5 per cent, malignant involvement of the cardia was recognized by gastroscopy alone. Of the 6 cases with a primary lesion, 5 were diagnosed by gastroscopy only. (The reviewer noted that a stomach lesion had been suspected by a radiologist in 3 of these 5 cases. In 1 of the other 2 cases the radiologist did not feel that a neoplasm was ruled out.) In 5 cases secondary extension of lesions to the cardia was not evident upon roentgen examination.

The author emphasizes that if obstruction is encountered at the cardia during gastroscopy, further investigation is necessary before concluding that such obstruction is due to cardiospasm.

An accurate preoperative recognition of the location and extent of gastric cancer is essential to proper treatment, particularly if total gastrectomy is to be performed effectively.

Three illustrations, including 1 roentgenogram; 5 tables. C. R. PERRYMAN, M.D.
Baton Rouge, La.

Idiopathic Megaduodenum: Report of a Case. J. E. McClenahan and Bernard Fisher. Am. J. Digest. Dis. **15:** 414–416, December 1948.

A dilated duodenum may be caused by congenital bands, aberrant vessels, or faulty rotation. It may also be secondary to adhesions from cholecystitis or operation, carcinoma, tuberculosis, or syphilis. Obstruction by tumors may also cause dilatation. In the case reported here, no cause for the condition was found.

A twenty-three-year-old white male complained of feeling tired and weak. He had been having tarry stools for some time, and on one occasion had vomited blood. X-ray examination revealed a markedly dilated duodenum with an ulcer just distal to the pylorus. Operation was performed and the roentgen observations were confirmed.

This was the only case of idiopathic megaduodenum seen in the last twenty-five years among 250,000 admissions and 3,500 autopsies.

Two roentgenograms. JOSEPH T. DANZER, M D.
Oil City, Penna.

Pre-Operatively Diagnosed Ileus Due to Gall Stone. Egan Norinder and Roland Gay. Acta radiol. **30:** 479–491, Dec. 31, 1948.

The authors add 5 cases of preoperatively diagnosed gallstone ileus to the literature. Two cases presented the symptom triad of ileus, fistula, and stone in the bowel on the plain film, so that no further roentgen examination was necessary. Three cases showed none of these signs in plain films but were diagnosed with the aid of a small contrast meal.

Air in the biliary tree is not in itself pathognomonic of gallstone ileus, even in the presence of an ileus.

Nothing new is added by this paper, but the material is well presented and the illustrations are informative.

Nine illustrations. M. M. MANALAN, M.D.
Indiana University

Polypoid Lesions of the Colon and Rectum. Robert A. Scarborough and Russell R. Klein. Am J. Surg. **76:** 723–727, December 1948.

Three important questions arise upon the discovery of any polypoid lesion of the colon and rectum: (1) Is this a single lesion or is it a manifestation of a pathologic process which also involves some other portion of the colon? (2) Is the lesion benign, premalignant, or malignant? (3) What is the proper treatment? The authors discuss these problems based upon their experience in a series of 458 patients in whom the original clinical investigation revealed presumably benign polypoid disease.

The incidence of polyps in males in the present series was 10 per cent greater than in females. The age of the patients ranged from five days to ninety-four years. Sixty-four per cent of the patients were found to have a single polyp, 31 per cent had from two to ten polyps, and 5 per cent had diffuse polyposis of the colon. The diagnosis of a single polyp was accepted only after thorough investigation by barium and an air-contrast examination following proctoscopy.

The problems of roentgen diagnosis of polypoid lesions beyond reach of the proctoscope are emphasized. Barium enema examination has a diagnostic accuracy of about 90 per cent in well advanced carcinoma of the colon. In demonstration of benign polyps this figure is greatly reduced, even with the valuable adjunct of air-contrast examination. Often two or more examinations are necessary before it is possible to show the lesion responsible for gross blood in the stool.

The presence of gross blood in the stool is frequently attributed to diverticulitis of the colon, and demonstration of diverticula by barium enema is commonly considered adequate evidence for this conclusion when sigmoidoscopic and x-ray examination have not disclosed the presence of ulcerative disease or carcinoma. While it is true that bleeding can occur in diverticulitis, in the authors' experience such bleeding usually has been found to be due to a coexisting adenomatous neoplasm. In 16 patients with a previous diagnosis of diverticulitis as the cause of rectal bleeding, removal of polypoid tumors of the colon brought an end to the bleeding although diverticular disease was still present.

Differential diagnosis is largely dependent upon roentgen examination of the colon. The presence of diverticula, and particularly distortions resulting from diverticulitis, greatly increase the difficulty of demonstration of polypoid lesions. This is sometimes impossible even with repeated examinations and use of the air-contrast technic. In such instances the continued

evidence of blood coming from above the reach of a proctoscope is sufficient indication for exploratory laparotomy and thorough search for a probable polypoid tumor.

Proper treatment is dependent upon accurate determination of the presence or absence of malignancy. Pathologic examination of the entire polyp is desirable for accurate diagnosis. In this series, excluding cases of diffuse polyposis, 211 polyps in 112 patients were destroyed by electrodesiccation through a proctoscope; 326 polyps in 182 patients were removed by diathermy snare and examined microscopically. Thirteen of these were found by microscopic examination to have undergone definite malignant change. Of these 13, 9 were submitted to radical resection, and in 3 residual carcinoma was demonstrated in the perirectal tissue or regional lymph nodes. In 4 patients local excision of adenomatous polyps with small areas of malignant change and long pedicles was carried out. Single grossly benign polyps were removed by colotomy in 37 patients; 12 of these 37 polyps showed definite malignant change on microscopic examination.

Removal of a polyp or resection of a carcinoma does not end the responsibility of the surgeon. Periodic sigmoidoscopic examination should be done and roentgen studies should be made whenever signs or symptoms suggest the possibility of a new lesion.

Acute Obstruction of the Colon with Special Reference to Factors of Mortality. Marshall L. Michel and Emit L. McCafferty. Arch. Surg. **57:** 774–788, December 1948.

Acute obstruction of the large bowel is a rather uncommon, highly fatal form of intestinal obstruction. The high mortality is due in part to the poor condition of many of the patients, whether from advanced age or from the chronic primary disease, often cancer. But another basic reason for this high mortality is the failure to distinguish the condition from acute small bowel obstruction, which has a very different therapeusis. This distinction can be made by the more frequent and accurate use of roentgenography, particularly following a barium enema. A flat plate alone seldom locates the obstruction, while a barium enema study usually does: this was in fact the case in 32 of 37 patients in whom the enema examination was used by the authors. Barium by mouth is contraindicated.

Conservative and temporizing measures are highly dangerous; the surgical attack should not be delayed. Except in the case of volvulus, it is better not to touch the causative lesion at the initial operation; indicated treatment of this should be deferred until recovery from the obstructed state.

Six charts. LEWIS G. JACOBS, M.D.
Oakland, Calif.

Traumatic Rupture of the Choledochus, Associated with an Acute Hemorrhagic Pancreatitis and a Bile Peritonitis. N. Frederick Hicken and Vernon L. Stevenson. Ann. Surg. **128:** 1178–1183, December 1948.

An interesting case report of a ruptured common duct in a 7-year-old boy is presented. The lesion occurred when he received a crushing injury to the upper abdomen, with compression of the impinged viscera against the lumbar spine. A week later he was acutely ill, with severe paraumbilical pain, nausea, vomiting, progressive jaundice, and temperature of 104.8° F. There were marked tachycardia and tachypnea, bilateral pleural effusion, ascites, and intense acidosis. Emergency supportive measures were instituted, and abdominal paracentesis recovered 3,000 c.c. of clear golden bile, which was sterile. Laboratory findings showed an anemia, leukocytosis, acholic stools, and choluria.

Following intensive preoperative care, laparotomy was done; 2,300 c.c. of normal bile being aspirated from the peritoneal cavity and 2,100 c.c. from the lesser omental sac. A cholangiogram made with the patient on the operating table revealed a complete block at the ampulla of Vater, with a trickle of diodrast through a laceration on the posteromedial wall of the common bile duct. It was felt that the obstruction was due to traumatic edema of the pancreas. A large rubber catheter was inserted into the gallbladder to decompress the biliary tree, and a Penrose drain was placed in the lesser omental bursa. The laceration in the common duct was not disturbed.

A postoperative cholangiogram a week later showed persistent obstruction of the ampulla, but no leak from the common duct. A third study, a week later, revealed relief from the obstruction, and decompression was discontinued. Recovery was uneventful.

The authors conclude that lacerations of the choledochal wall will heal spontaneously providing the extrahepatic ductal system is kept decompressed by external drainage. Progress of healing and return of normal function are determined by serial cholangiograms.

One drawing, two roentgenograms.
EDWARD E. LEVINE, M.D.
Dearborn, Mich.

Extensive Visceral Diverticulosis. David Fiske and Leonard M. Asher. J. A. M. A. **138:** 1153–1155, Dec. 18, 1948.

Diverticulosis involving a hollow viscus is not an uncommon medical observation, but the existence of multiple diverticula of numerous viscera is infrequent. The authors' patient, a 52-year-old white man, had a diverticulosis involving the stomach, duodenum, jejunum, ileum, urinary bladder, and urethra, but without diverticulosis of the colon, which is the hollow organ most frequently affected by this condition. Associated with the diverticulosis were multiple abdominal wall hernias.

Theories of the formation of diverticula are briefly discussed, and literature on their occurrence is reviewed. This case is thought to represent a clinical entity based upon weakness of both smooth and striated muscle.

Three roentgenograms. M. M. FIGLEY, M.D.
University of Michigan

THE MUSCULOSKELETAL SYSTEM

Polyostotic Fibrous Dysplasia of Bone. H. Brooke. Canad. M. A. J. **59:** 555–557, December 1948.

The author presents two cases of fibrous dysplasia of bone and discusses the diagnosis and differential diagnosis of this relatively new entity with which the radiologist is being more frequently confronted.

The radiographic findings must be considered in the light of the clinical information at hand "and then only should we venture on anythi. g other than a di-

ferential diagnosis." The cystic appearance of the bony lesions most closely simulates osteitis fibrosa cystica, which is usually differentiated by means of calcium and phosphate blood levels. The slow progression of the process, usually with a history of injury, as well as the precocious menstruation and hyperpigmentation mentioned by Albright as occurring in severe forms of the disease in children, further aid in its identification.

The appearance of a biopsy specimen is fairly characteristic, showing a replacement of normal bony architecture by fibrous connective tissue and osteoid tissue, with both osteoblasts and osteoclasts. Histologically, the condition is frequently confused with ossifying fibroma.

Polyostotic fibrous dysplasia has a much brighter prognosis, fortunately, than most of the conditions with which it is confused and, for this reason, it is well to keep it in mind as a possibility.

Six roentgenograms. ROBERT H. LEAMING, M.D.
Jefferson Medical College

Ollier's Dyschondroplasia. Thomas G. Potterfield and Juan Gonzalez. J. Pediat. **33:** 705–709, December 1948.

A case of Ollier's dyschondroplasia is reported in a three-year-old girl. The humerus, radius, femur, tibia, scapula, os pubis, first metatarsal, and phalanges on the right were involved. The left side was not affected.

The x-ray appearance was that of non-ossified areas of rarefaction alternating with areas of normal bone. These non-ossified areas were arranged both longitudinally and obliquely in relation to the shaft and involved both the cortex and medullary space.

The disease is the result of failure of ossification of the cartilage in the diaphysis. Non-ossified cartilage may proliferate normally but occasionally may undergo independent growth, with the formation of large tumors and attendant deformity. Since the condition often occurs close to the epiphysis, it frequently causes disturbances in growth.

The only known treatment of value is proper orthopedic management of the resulting deformities.

Eight roentgenograms. EUGENE P. KUTZ, M.D.
Baltimore (Md.) City Hospitals

Roentgenologic Picture of Congenital Syphilis, Especially in Infancy. P. Flemming Møller. Acta radiol. **30:** 443–456, Dec. 31, 1948.

The roentgenologic manifestations of syphilis in early infancy are stressed, with occasional reference, for comparison, to acquired or late congenital lesions. In both acquired and congenital syphilis, periostitis, gummatous changes, hyperostosis, or osteitis may be present. Osteitis is usually seen in the late forms of the congenital disease. It should be remembered that the osseous lesions of congenital syphilis may be delayed in appearing until puberty or even as late as the twenty-eighth year.

In early infancy, the bone involvement is most often localized in the growth zones, in the form of the so-called syphilitic osteochondritis. The deficient osteoblastic activity just below the provisional line of calcification and the formation of a soft granulomatous tissue here make this metaphyseal region weak and therefore subject to occasional fracture, with the production of Parrot's pseudoparalysis.

Three stages of osteochondritis are noted: (1) the initial stage with a more or less dense and slightly frayed zone of calcification; (2) a stage in which the metaphyseal border is still more dense, with an increase in the ragged appearance and apparent separation from the diaphysis by a broad clear zone; (3) a final stage in which the metaphysis has become completely obliterated, leaving a defect in the diaphyseal end.

Periosteal changes may be seen in infancy but are usually noted in the second to third month or in late syphilis. Granulomatous changes also may occur in the newborn but are more common in the second to third month or later, often developing into syphilitic osteomyelitis.

The hyperostosis of late syphilis may occasionally be confused with sclerotic osteomyelitis. One such case is reported in a boy fourteen years old.

The differential diagnosis of congenital syphilis in infancy, scurvy, rickets, and acute osteomyelitis is discussed.

The clinical picture is often very different from the roentgenologic. The cutaneous and visceral lesions of early syphilis may be slight or absent, while the osseous manifestations are usually characteristic, making possible a roentgenologic diagnosis.

Nineteen roentgenograms. J. SCOTT, M.D.
Indiana University

Myelography by Resorbable Contrast Substances. J. Th. van der Werff. Acta radiol. **30:** 493–498, Dec. 31, 1948.

Myelography was done with 10 c.c. of various resorbable contrast substances under spinal anesthesia—20 per cent abrodil, 25 per cent dijodon, 37.5 per cent uropac, 37.5 per cent uroselectan B. Four exposures (in lateral, prone, and two oblique positions) were made, with the use of a tilting table and a horizontal beam. The author believes that the exposure with the patient prone is especially valuable, as it furnishes a "complete insight into the width of the epidural space."

After the procedure, 20 c.c. of spinal fluid and contrast substance was removed and the patient was put to bed with instructions not to lie on his back or healthy side for twenty-four hours. This seemed to cut down the number of complications.

Of 60 patients thus examined, 29 showed disk protrusion which was confirmed at operation. Five others showed protrusion but for various reasons were not operated upon. In 2 cases multiple disk prolapses were demonstrable roentgenographically, but only a single one was found at surgery. These were explained as possible examples of intermittent prolapse. In the remainder of the series no abnormality was found and surgery was not undertaken.

Six roentgenograms; 2 drawings.

R. DATZMAN, M.D.
Indiana University

Absorption of Myelotrast (Abrodil) from the Spinal Canal. Haakon Ødegaard. Acta radiol. **30:** 464–469, Dec. 31, 1948.

The author uses 10 c.c. of 20 per cent myelotrast (abrodil) for myelography, with the patient under spinal anesthesia. This substance is heavier than spinal fluid and, though irritating, it is supposedly absorbed in a short time. A tilting table is used, and the patient lies in a sloping position with the upper part of the body elevated during the injection and the subsequent ex-

amination. Films were taken at intervals to check the rate of absorption. Contrary to reports that the medium is absorbed in thirty minutes, the author found that often an hour was needed for complete absorption. He makes it a practice, therefore, to keep the patient lying with the upper part of the body elevated for that length of time after examination.

Nine roentgenograms, illustrating 4 cases.

R. DATZMAN, M.D.
Indiana University

The Subarachnoid Spaces of the Root Sheaths in the Lumbar Region. K. Lindblom. Acta radiol. **30:** 419–426, Dec. 31, 1948.

The author studied 35 gross specimens of the dural sac with adjacent roots and ganglia. Four other specimens were given detailed study and myelograms from 150 cases were reviewed.

Roentgenographic and anatomic study of the subarachnoid space at the lumbar nerve roots showed that there are great variations. The subarachnoid space ends with small processes, distal to which there may be cysts and fissures filled with a clear colorless fluid. No communications were found between the main subarachnoid space and these cysts and fissures. Such bulges of the subarachnoid space, sometimes seen on myelograms, may be explained as normal variations of the shape of the dural sac.

Seventeen illustrations. O. R. RUSSELL, M.D.
Indiana University

Skeletal Lesions in Hodgkin's Disease. Ernest H. Falconer and Maurice E. Leonard. Ann. Int. Med. **29:** 1115–1131, December 1948.

A comparison of reported series of Hodgkin's disease in which skeletal lesions were determined roentgenologically and others which were studied at autopsy shows a considerably higher incidence of bone involvement in the latter. This the authors take to indicate that roentgenologic statistics may be misleading unless the limitations of the method are kept in mind. They believe that the incidence of skeletal involvement as shown roentgenologically is necessarily incomplete and too low, as marrows with Hodgkin's lymphogranuloma may contain lesions too small to involve the adjacent cortical portions of the bone.

Because of the general agreement that skeletal involvement in Hodgkin's disease means marrow involvement, a study was made of the aspirated sternal marrow in 59 patients with the disease in an attempt to accumulate data which might suggest invasion of the marrow. In the marrow from these cases a uniform cellular pattern was demonstrated, showing an increase of the myelocytic elements (a shift to the left) with emphasis on the neutrophilic and eosinophilic myelocytes, the band neutrophiles, eosinophilic segmented cells, and plasmacytes. This same cellular pattern was present in the aspirated sternal marrow of 12 patients of whom 10 showed marrow involvement at autopsy.

Twelve illustrations (8 roentgenograms); 2 tables.

GYNECOLOGY AND OBSTETRICS

A New Contrast Medium (Rayopake) for Hysterosalpingography. Albert Sharman. J. Obst. & Gynaec. Brit. Emp. **55:** 770–773, December 1948.

Following a brief historical review of the use of radiopaque substances in hysterosalpingography, the author reports his experience with rayopake, which has only recently been introduced into Europe.

Comparisons of viscosity and density with "iodatol" (almost the equivalent of heavy lipiodol) were made, and it was found that while rayopake was not so dense nor so viscous, its radiopacity was quite satisfactory for hysterosalpingography, though not for intravenous pyelography. The rapid absorption and the absence of the undesirable features of iodized oil are its chief advantage. In a series of 50 patients satisfactory results, with no ill effects, were obtained.

Twenty illustrations, including 19 roentgenograms.

S. F. THOMAS, M.D.
Palo Alto, Calif.

Acute Hydramnios. Paul F. Mueller. Am. J. Obst. & Gynec. **56:** 1069–1076, December 1948.

Among the first 49,793 deliveries at the New York Hospital there were 4 cases of acute hydramnios and 62 cases of chronic hydramnios. The fetal mortality was 100 per cent in the 4 acute cases and 52 per cent in the chronic cases. Of the 34 infants which did not survive, 19 had congenital abnormalities and 7 had erythroblastosis.

This series of cases and a study of the literature shows that there is a correlation between congenital anomalies and hydramnios. It is therefore felt that radiographic examination of the abdomen should be performed in all cases in which hydramnios is diagnosed. Anencephaly and other congenital anomalies are frequently demonstrable radiographically.

Four tables. JOHN DECARLO, JR., M.D.
Jefferson Medical College

Cystoscopy and Pyelography Following Paravesical Extraperitoneal Cesarean Section. Edith K. Mangone. Am. J. Obst. & Gynec. **56:** 1138–1141, December 1948.

Extraperitoneal cesarean section in the presence of potential or frank infection is regarded with varying favor. The author demonstrates that better understanding of the anatomy of the fascial or "areolar" tissues and improvement in technic (Norton: Am. J. Obst. & Gynec. **51:** 519, 1946) have lessened the validity of objections to its use.

Since the procedure is chiefly one of bladder dissection, cystoscopic and intravenous or retrograde pyelographic studies were made some three months postpartum in 18 patients to determine its effect on bladder function. Each of the patients was reported as having a normal bladder shadow, normal ureters both in size and anatomic relationship, and normal kidney pelves. The average time for excretion for each kidney was 3.3 minutes.

The author concludes that if bladder manipulation is carried out with the bladder empty, injury to this organ does not occur with careful dissection and that, following paravesical extraperitoneal cesarean section, there is no impairment of bladder function and no disturbance of bladder or ureteral anatomy.

ROBERT H. LEAMING, M.D.
Jefferson Medical College

THE GENITO-URINARY SYSTEM

Perinephritic Abscess: A Review of Twenty-Two Cases. Harry Bergman and Samuel Simon. Urol. & Cutan. Rev. **52:** 704–708, December 1948.

A review of 22 cases of perinephritic abscess is re-

ported: 10 were extrarenal in origin, 5 were secondary to renal disease, and 7 were of undetermined origin. *Staphylococcus aureus*, and occasionally *Staphylococcus albus*, were found in the extrarenal type; whereas, *E. coli* was most frequently encountered in the renal type. Three cases were bilateral.

Characteristic symptoms were loin and costovertebral angle pain, chills and fever, and psoas irritation manifested by hip flexion and difficulty in walking.

Roentgen findings included obliteration of the psoas shadow, scoliosis of the lumbar spine with convexity to the involved side, renal and diaphragmatic fixation, and renal displacement. Pathological kidney and ureteral changes were demonstrable in 9 cases. In one case of bilateral involvement, the psoas shadows were obliterated and the spine was straight.

Preoperative diagnosis was made in 15 cases, followed by excision and drainage plus chemotherapy.

MAURICE D. SACHS, M.D.
Cleveland, Ohio

Urethrography in Infants and Children. Lorye E. Hackworth. J. Urol. **60:** 947–951, December 1948.

Visco-rayopake has been used for urethrography in infants and children at Children's Hospital, Boston, Mass., since 1944. A modified formula, called simply rayopake, contains less iodine and gives a somewhat less dense shadow, thereby making it possible to discern small lesions which would be obscured by a denser shadow. This is miscible with water or urine in all proportions.

A 10-c.c. hypodermic syringe is used, provided with a cystoscope nipple. This is introduced into the external meatus and 3 to 10 c.c. of visco-rayopake are injected slowly and steadily. Exposures are usually made in the anterior-posterior and right oblique positions. If a voiding urethrogram is desired, the visco-rayopake may be injected into the bladder, where it mixes with the bladder urine.

Seventeen roentgenograms of normal and pathologic conditions.

HORACE G. BUTLER, M.D.
University of Pennsylvania

Urethral Diverticulum with Calculi in Women. Harry A. Zide. California Med. **69:** 458–461, December 1948.

Calculi in a urethral diverticulum in the female are rare. The diverticula are usually attributable to infection of urethral glands, with subsequent periurethral abscess and sac formation. Calculus formation within the diverticulum is believed to be due to infection of urine, with stagnation and deposition of urinary salts. Symptoms include a vaginal mass, ureteral or perineal pain, pain on intercourse, sometimes hematuria, especially after coitus, and evidence of urinary tract infection of ascending type.

Diagnosis can be made on palpation of a mass on the ventral surface of the urethra. A roentgenogram will reveal a calculus in the subpubic region. Studies with the aid of an opaque medium demonstrate the diverticulum. The orifice to the diverticulum may be demonstrated by means of urethrocystoscopy, either with a right angle lens or the foroblique lens, while viewing the ventral surface of the urethra. Treatment consists of surgical excision of the sac and calculi.

A case is reported.

Three roentgenograms. MAURICE D. SACHS, M.D.
Cleveland, Ohio

SCLERODERMA

Roentgenologic Manifestations of Scleroderma. David G. Pugh. Am. J. M. Sc. **216:** 571–580, November 1948.

Scleroderma is a connective-tissue disease, fundamentally a collagen disturbance. Induration of the tissue is due to collagen-fiber thickening and proliferation. The changes are not ascribable to vascular lesions. Visceral involvement, especially in the type known as acrosclerosis, occurs fairly frequently and may be observed roentgenologically.

In the esophagus scleroderma is manifested by weakened, incomplete, or absent peristalsis, slow transit time, tendency to dilatation and "gaping" or failure to collapse, narrowing of the distal third, and occasionally patulousness of the phrenic portion, permitting gastric regurgitation. Complete obstruction does not occur. Shortening of the esophagus with diaphragmatic hernia has been reported, an observation which, with others, indicates that the disease leads to stiffening and patulousness, to regurgitation and chronic esophagitis, and eventually to fibrosis, shortening, and herniation. Endoscopy confirms the inflammatory changes, revealing granulation tissue, mucosal loss, cicatricial stenosis, and induration. The author suggests that chronic inflammation and its sequelae may be responsible for much of the change in necropsy specimens (mucosal loss, submucosal sclerosis, muscle atrophy and fibrosis).

Less frequently reported are changes of scleroderma lower in the gastro-intestinal tract. Delay in gastric empyting, impaired peristalsis in the upper small bowel with delayed transit, localized dilatation, segmentation, and even generalized dilatation have been reported. Goetz described atrophy, cellular infiltration and fibrosis of muscle and prominence of neuromuscular apparatus in necropsy specimens.

A characteristic diffuse fibrosis in the lungs, sparing the apex and lateral base, has been described in pulmonary scleroderma. Histologically, the elastic fibers in the alveolar walls are replaced with collagen, and cyst-like spaces develop from alveolar breakdown. Peribronchovascular fibrosis is also noted.

In scleroderma of the heart, Weiss described enlargement, poor pulsations, and a triangular shape, clinically, with pathological findings of focal connective-tissue proliferation unrelated to blood vessels. Various rhythmic disorders were noted. Goetz observed a small heart of normal shape but with diminished pulsations.

A widened periodontal space has been observed in dental films of patients with scleroderma, particularly about the posterior teeth. The widening includes the roots and is distinguishable from other disorders of the pericementum by its uniformity. Actual thickening of the periodontal membrane to two to four times the normal has been directly observed. Looseness of involved teeth is not notable.

In advanced acrosclerosis, distal phalangeal resorption occurs. Increased phalangeal density and synostosis of the distal two phalanges may be seen. Sand-like or plaque-like calcinosis, mostly about pressure points, such as fingers, elbows, and ischial tuberosities, is also observed.

Several organs may be involved in the same patient.

This paper is essentially a review, and a good bibliography is included.

JAMES ALLAN READ, M.D.
The Henry Ford Hospital

RADIOTHERAPY

Cancer of the Lower Lip. Survey of the End Results of Treatment; All Cases Treated at the Brooklyn Cancer Institute, 1930 through 1944. William E. Howes and Joseph Rosenstein. Am. J. Roentgenol. **60:** 763–775, December 1948.

On the basis of 112 cases treated between 1933 and 1944, and proved pathologically by biopsy, the authors discuss the racial and hereditary tendencies, etiology, pathology, diagnosis, and treatment of carcinoma of the lip. Many of the lesions, when first seen, measured over 2 cm. in diameter. Eleven of the patients (10 per cent of the series) had palpable submental, submaxillary, or cervical lymph node involvement at the first visit. In 21 (18 per cent) palpable nodes appeared after treatment was begun.

These 112 cases were treated by surgery, irradiation, or both. In only 6 cases was surgery the only treatment. Five of the number survived five years or longer.

The following forms of irradiation were used:

(1) *Superficial Roentgen Therapy:* Prior to 1936, superficial therapy was employed (80 to 135 kv., h.v.l. 1.0 to 3.0 mm. aluminum). A single dose of 4,000 to 6,000 r in air was delivered. In 6 of the 11 patients so treated, metastases developed. These cases were considered failures.

(2) *Deep Roentgen Therapy* (200 kv., Thoraeus filter, h.v.l. 1.8 mm. copper, 30 cm. target-skin distance, 600 r in air daily): A total air dose of 7,200 r within two weeks was attempted. With this dose and technic, no necrosis or any failure of the intense radiation reaction to heal was observed. Of the 44 cases so treated, 42 were apparently cured. In 2 patients cervical metastases later developed and were radically resected. In other words, with this type of therapy no failures were recorded. Recurrences were observed when the dose was reduced to 4,500 r in air or less.

(3) *Contact Roentgen Therapy* (air-cooled; 45 kv., 2 ma., 2.1 cm. target-skin distance; h.v.l. 3.0 mm. aluminum): A dose of 15,000 r in air was delivered in one sitting to 3 patients with lesions measuring more than 1.0 cm. in diameter. None had evidence of recurrence in a follow-up interval of six years. A fourth patient was given a larger dose because the lesion was more extensive. He died with metastases four years after treatment.

(4) *Radium Mold:* Radium element tubes with 1.0 mm. platinum filter were arranged in a wax mold so that the skin distance was 1.0 cm. According to the extent of the lesion, 1,200 to 2,500 mg. hr. were delivered. After 1939 the computed tumor dose was 6,000 gamma roentgens. Of the 10 patients thus treated, 3 had lymph node extension at the time of treatment or later. In one of these the disease was controlled for ten years by interstitial radium and radon; the others died within two years. Seven patients, when last seen, had been free of disease for two to sixteen years.

(5) *Interstitial Radium:* Radium needles or radon seeds were inserted in and about the lesion in 13 cases for a minimal dose calculated at 6,000 gamma roentgens. All 13 patients had survived for a period averaging five to six years [but this does not mean that each case represents a five-year survival].

When any one of these foregoing methods failed, combined procedures were substituted. Twenty-four cases had combined therapy as follows:

1. Surgery and roentgen therapy, 6 patients.
2. Interstitial radium and surgery, 2 patients.
3. Surgery, roentgen irradiation, and radium, 1 patient.
4. Radium and roentgen therapy, 12 patients. In 5 of these a satisfactory end-result was obtained. Six (50 per cent) in whom cervical metastases developed are listed as unsatisfactory.

Cervical metastases were treated either by radon seed implantation or radical neck dissection, and the authors favor the latter method. Of the 8 cases that had known cervical lymph nodes on admission, only 1 had a satisfactory end-result. In this case roentgen therapy to the lip lesion was followed by a radical neck dissection. Of the 21 cases (18 per cent) in which metastases developed after the onset of treatment, only 4 gave a satisfactory end-result, 2 treated by radon seed implantation and 2 by radical neck dissection.

Eighty-six of the 112 cases (77 per cent) have been followed for a period of two to sixteen years and are considered to have satisfactory end-results.

A table summarizes the pertinent data in every treated case.

Nine figures, including before-and-after photographs in 6 cases.

SAMUEL MORCHAN, M.D.
Indiana University

Treatment of Advanced Cancer Involving the Ear. Charles L. Martin and James A. Martin. Am. J. Roentgenol. **60:** 750–757, December 1948.

The authors agree with Pfahler and Vastine that "a greater percentage of failures occur in the treatment of cancer in the region of the ear than in any other relatively superficial portion of the body."

By various combinations of electrosurgery, roentgen, and radium therapy the authors were able to salvage 50 per cent of 35 advanced cases, many of which had previously been treated unsuccessfully.

All cartilage showing malignant invasion or irradiation necrosis is excised electrosurgically under local anesthesia before starting irradiation. When possible, a portion of the upper auricle is left so that glasses may be worn with ease. Immediately following surgery, irradiation is instituted by either roentgen rays or interstitial radium. Good results have been obtained with twelve daily doses of 315 r in a period of two weeks through a 10 × 10-cm. field, using 200 kv., a target-skin distance of 50 cm., and filtration of 0.5 mm. copper and 1.0 mm. aluminum. The total air dose amounts to 3,780 r but, allowing for the time factor, the effective tumor dose at 2.0 cm. becomes 2,176 r with back-scatter, or 3.2 T.E.D.

The alternate technic consists of a grid implantation of low intensity radium needles carrying 0.6 to 0.8 mg. per linear centimeter of active length, for 168 hours. A total dosage of 6,000 to 12,000 gamma roentgens is attained.

Metastatic lymph nodes are treated by interstitial radium as in the primary lesion plus six daily doses of 350 r of x-ray with a high-voltage machine.

Nineteen photographs; 1 diagram.

J. A. CAMPBELL, M.D.
Indiana University

Malignant Tumors of the Cheek in Children. Report of Two Cases. Grant E. Ward and E. Roderick Shipley. Am. J. Roentgenol. **60:** 758–762, December 1948.

Malignant tumors of the face are 100 times less common in children than in adults according to Martin (J. Pediat. **15:** 363, 1939), and tumors located in the cheek are very rare. The authors describe two cases.

The first patient was a four-year-old colored girl, and a biopsy specimen was interpreted as anaplastic malignant tumor, possibly metastatic from an adrenal neuroblastoma. The second patient was an eleven-months-old white girl. The first biopsy specimen in this case was read as an embryonic carcinoma and a second specimen, taken four months later, was regarded as "low-grade malignant tumor arising from aberrant salivary tissue."

The first patient was treated one month after appearance of the tumor by interstitial low-content radium needles, for a necrotizing dose of 2,993 mg. hr., which resulted in considerable deformity of the eyelid and upper lip and probable radiation cataract, but with a five-year cure and a fair cosmetic result. The second patient was treated by surgical excision followed immediately by 375 mg. hr. of radium in the wound, and then by roentgen therapy, 1,800 r (200 kv., 0.5 mm. copper filter) given in ten days through a 3-cm. portal. In four months there was a recurrence above the surgical scar, and surgery, rather than radiation, was again attempted because of the likelihood of damage to the adjacent eye and the probability of radioresistance. The surgeon considered the tumor inoperable at this time.

From reported statistical evidence, the authors conclude that sarcoma is twenty-two times as frequent as carcinoma in persons under twenty-five years of age. They discuss the occurrence of salivary-gland tumors in children and call attention to the possible presence of normal salivary gland tissue in the cheek.

Two photographs; two photomicrographs.

W. E. CHILDS, M.D.
Indiana University

Cancer of the Larynx. Five Year End Results in a Series of Patients Treated Between 1930 and 1942. Chevalier L. Jackson, John V. Blady, Charles M. Norris, and Walter H. Maloney. J. A. M. A. **138:** 1080–1082, Dec. 11, 1948.

The authors describe their findings in 612 patients with cancer of the larynx treated at Temple University Hospital from 1930 to 1947, inclusive. Surgical treatment was used in 453 cases (laryngofissure or partial laryngectomy in 209 and total laryngectomy in 244), and irradiation in 159. Five-year end-results can now be computed on only 371 patients, through December 1942. Of these, 118 had laryngofissures, 102 had laryngectomies, and 91 had initial courses of treatment by irradiation.

Because of death from other causes or failure of follow up, only 102 of the 118 laryngofissure cases were included in the five-year survivals. Of the 102 laryngectomies, 123 were done prior to 1940 and were not done by the author. Four patients died of other causes or were lost to follow-up, leaving 35 "determinate" cases. Three of the group had previous laryngofissures and 1 had irradiation. Of the 91 patients treated by irradiation, 83 were "determinate." Thus, the series treated by the authors from 1930 to 1942, inclusive, includes 216 patients with a final survival rate of 72 per cent.

Of the 19 recurrences in the 102 patients treated by laryngofissures, 74 per cent occurred in the first three years and 4, or 22 per cent, after five years. There was an initial cure rate of 81 per cent and a salvage of 51 per cent of the recurrences in the laryngofissure group.

A 66 per cent survival rate was reported for the total laryngectomie. Sixty-nine patients without palpable nodes who were treated with irradiation had a 51 per cent five-year survival rate. Fourteen with node involvement did not survive five years. Cases treated with irradiation included both intrinsic and extrinsic cancer.

Recurrences after laryngectomy in the authors' series were not successfully treated. Recurrences after irradiation were salvaged by second courses of irradiation and laryngectomy.

Six tables; 1 chart.

S. B. FEINBERG, M.D.
University of Michigan

Carcinoma of the Breast. Stanford Cade. J. A. M. A. **138:** 1083–1086, Dec. 11, 1948.

Because of the increasing number of deaths due to breast cancer, the author reviewed the present day methods of treatment and critically analyzed the causes of failure to reduce the mortality rate.

Smaller families, failure of lactation, and longevity contribute their share in the increase of the disease.

The expectation of life in untreated patients has been computed to have a mean duration of 39.3 months. It is therefore essential that one should not rely on three-year survival rates.

The author assesses the state of the patient on the basis of age, clinical type and histologic variety of tumor, duration of symptoms, physiologic state of the breast, and extent of the disease. To assess the immediate and ultimate prognosis, to estimate the probable expectation of life and, above all, to find out the value of any given method of treatment, clinical staging is of paramount importance.

The clinical staging of Portmann is used by the author. Stage I, tumor of the breast only; Stage II, tumor of the breast and cutaneous changes and/or axillary nodes; Stage III, tumor of the breast and supraclavicular or contralateral axillary nodes or fixation to pectoral fascia; Stage IV, skeletal or visceral metastases.

In Stage I the method of choice is radical mastectomy. It is not contended that radiation alone cannot achieve total regression of the tumor for five to eight years; but at present it does not do so as certainly and as frequently as radical mastectomy. Nearly 90 per cent of the five-year survivals and 65 per cent of the ten-year survivals have been obtained by radical surgical treatment in Stage I cases.

In Stage II lesions, the results from surgical treatment show a great drop. Only 30 per cent five-year survivals are achieved. The ten-year survival rate is raised from 25 per cent by surgical treatment alone to 32 per cent by surgical treatment and postoperative radiation.

In Stage III cases, radiation alone has given better results than surgical treatment and is the method of choice. Here the ten-year survival rate for surgical treatment is 7 per cent whereas it is 13 per cent for radiation alone. A combination gives only 9 per cent.

Preoperative radiotherapy aims at sterilization of an

active neoplasm in the breast, of the skin over it, and of the immediate regional lymph nodes in the axilla and below the clavicle. At Westminster Hospital, in 448 cases analyzed between 1930 and 1942, the five-year survival rate with preoperative radiation was greater than with postoperative treatment, 73 per cent in the former and 53 per cent in the latter.

Postoperative radiotherapy is not indicated in Stage I cases, but is of value in Stage II.

Two tables.

S. B. FEINBERG, M.D.
University of Michigan

Surgical and Radiation Treatment of Cancer of the Breast. Rolf Bull Engelstad. Am. J. Roentgenol. **60:** 776–787, December 1948.

The author presents a statistical study of 1,384 patients treated or examined for cancer of the breast at the Norwegian Radium Hospital between 1932 and 1942; only patients followed five years are included in the study. He believes that radiation for cancer of the breast should be given postoperatively in one treatment period and with large doses. Removal of the overlying breast tissue makes possible a more homogeneous irradiation. The deep lymphatics which penetrate the chest wall are made more accessible, and the needless delay in operation involved in an attempt at preliminary irradiation of a possibly radioresistant tumor is avoided.

If sufficient radium is available, this type of irradiation is considered better than roentgen therapy. With either method, the routine fields are irradiated. If radium is used, a mold is made with a radium-skin distance of 3 cm., and a total of 3,500 to 4,000 gamma roentgens is delivered in six to seven days. With roentgen irradiation the factors are 175 kv., 4 ma., filter 1 mm. copper, focal skin distance 50 cm. Daily doses range from 350 to 400 r, with a total dosage of about 3,500 r per field. The advantage of radium is believed to be in a more favorable distribution of depth dose and the continuous irradiation with low intensity over a long period of time.

The cases are classified in four stages; Stage I, operable cancer of the breast with no clinically or histopathologically demonstrable spread; Stage II, operable cancer of the breast with clinically or histopathologically demonstrable operable metastases in the axillary lymph nodes; Stage III, inoperable cancer of the breast; Stage IV, inoperable cancer of the breast with distant metastases or recurrences. For statistical evaluation, Stages I and II are placed together, as those cases which receive the radical operation followed by irradiation. Several tables are included in an attempt to correlate social, physical, and chronological data with respect to the incidence of carcinoma in this series. Twenty per cent of the cases were Stage I, 35 per cent Stage II, 13 per cent Stage III, and 31 per cent Stage IV. Out of the 1,384 cases, only 7 patients have been lost to follow-up. Considering the four stages collectively, 37.8 per cent of the patients were symptom-free after five years. Of those with lesions of Stage I and II, i.e., operable lesions with or without axillary metastases, treated by operation and irradiation, 62.6 per cent were symptom-free at the end of five years. The highest percentages of symptom-free cases were among those receiving postoperative irradiation, 69 per cent of 609. Only a few cases received preoperative or preoperative plus postoperative irradiation.

The author feels that the prognosis of patients in the younger age group should not be particularly bad and that lactation or pregnancy should not carry the severe prognosis which it sometimes does in any respect except by virtue of the difficulty in diagnosis which occurs with the breast engorgement. He is not convinced of the benefit to be derived from preclimacteric castration of patients with cancer of the breast and reports on only a small series of cases treated with testosterone propionate.

It is not felt that radiation treatment will ever replace the radical operation but that the two should be used together for the best results. Large doses should be used and the dose given in each individual case should be just short of that which would produce any serious radiation injury. Since the fate of these patients is usually foretold by the presence of distant metastasis, an effort to prevent such formation should be the main task of the radiologist.

Sixteen tables.

JOHN SCOTT, M.D.
Indiana University

The Desirable Management of Fundal Carcinoma. Lewis C. Scheffey and Warren R. Lang. S. Clin. North America **28:** 1425–1444, December 1948.

The desirable management of any malignant lesion is its elimination in such a way that recurrence is unlikely and the patient's survival assured. This is a difficult goal to attain.

The purpose of this paper is "to discuss some factors relating to the etiology, pathology, symptomatology and diagnosis of fundal cancer which have a distinct bearing upon management, selective treatment and results." Histologic classification is confined to low, intermediate, and high grades of malignancy. The clinical grouping of the American College of Surgeons is followed.

The procedure adopted today in most gynecologic clinics is preliminary irradiation followed by surgery. Irradiation tends to promote local devitalization and attenuation, if not complete destruction of the growth, reduction of infection and sealing of the lymphatics, thus lessening the possibility of cancerous dissemination when surgery is employed later on, and finally reduction of the incidence of recurrent growth in the vaginal vault scar.

The authors quote various papers giving a considerable range of five-year cures, with surgery alone, irradiation alone, and various combinations of irradiation and surgery. They also bring up to date their own figures, supplementing an earlier report (Am. J. Obst. & Gynec. **52:** 529, 1946. Abst. in Radiology **49:** 263, 1947). Of 138 patients eligible to date (1921–1948) for five-year statistics, 77, or 55.8 per cent, have lived from five to twenty-five years. The authors' "planned technic" was used in 23 patients, of whom 21 survived from five to sixteen years. This technic of irradiation followed by resection, though permitting certain variations, consists most commonly in radium implantation followed in six or ten weeks by total hysterectomy and salpingo-oophorectomy.

A "poor risk" patient is usually treated with radium alone. The authors add the statement that they "are coming to realize more and more, however, that even the substandard patient may eventually warrant surgery either because of revised judgment or as a result of necessity."

Four illustrations.

EDSEL S. REED, M.D.
University of Louisville

Role of Surgery in the Treatment of Carcinoma of the Cervix. Charles D. Read. Am. J. Obst. & Gynec. **56:** 1021–1029, December 1948.

The Wertheim operation was generally abandoned with the advent of radiotherapy, but it is still done in selected cases at the author's hospital (Chelsea Hospital for Women, London). One of the most prominent advocates of the Wertheim operation in the author's experience is Victor Bonney, who up to 1936 had done 500 of these procedures, with an operability rate of 63 per cent of all cases seen, and with an over-all operative mortality rate of 14 per cent. His cases included Stages I, II, and III growths. Metastatic regional node involvement was present in 40 per cent of all cases. The five-year cure rate was 58 per cent in cases without lymph node involvement, and 23 per cent in cases showing positive node biopsy. The absolute cure rate is 25 per cent. In none of the cases was radiotherapy employed routinely either pre- or postoperatively.

In comparison, the author cites the results of five prominent workers in radiotherapy, covering 2,583 cases treated up to 1926. There was a five-year survival rate of 23.8 per cent. Later work has shown a substantial improvement over this figure, the results published in 1942 by the Marie Curie Hospital showing an 80 per cent five-year absolute survival rate in Stage I, 61.5 per cent in Stage II, 31.4 per cent in Stage III, and 6.0 in Stage IV.

At the Chelsea Hospital, the Wertheim operation has been performed 96 times, with an operative mortality of 8.3 per cent. The operation rate on patients seen has ranged from 9 to 20 per cent. The indications for this procedure, as practised there, are as follows:

1. Radioresistant growths proved either clinically or cytologically.
2. Columnar-cell carcinoma of the cervix.
3. Stenosis of the vaginal vault.
4. Complicating large fibroids or ovarian cysts.
5. Complicating salpingitis.
6. Refusal of radiation by patient.
7. Complicating pregnancy.

In conclusion, the author states that he advocates radical surgery in certain suitable cases rather than radiotherapy alone, and believes that this type of treatment has been badly underrated. However, he continues to favor radiotherapy in the average case as the treatment of choice for carcinoma of the cervix. He believes, also, that more extensive practice of lymphadenectomy might lead to an improvement in results.

Eight tables. HARVEY J. THOMPSON, JR., M.D.
Jefferson Medical College

Controversial Factors in the Management of Carcinoma of the Cervix. Robert A. Kimbrough, Jr., and Craig W. Muckle. S. Clin. North America **28:** 1415–1423, December 1948.

An interesting historical survey of the treatment of carcinoma of the cervix is presented, including treatment from 1560, when Andrea della Croce of Venice performed a hysterectomy with a cautery iron for carcinoma in a prolapsed uterus, until the present time.

The authors describe their own technic of radiation therapy and discuss some of the pertinent factors involved in the radical surgical approach. They use a combination of roentgen therapy and local radium application and report an absolute survival rate of 27.6 per cent among 130 patients thus treated from Jan. 1, 1933 to Dec. 31, 1941. In Stage I cases the salvage was 85.7 per cent; in Stage II, 47.3 per cent; in Stage III, 18.2 per cent. There were no five-year survivals in Stage IV cases.

It is concluded that whether early carcinoma of the cervix is treated by irradiation or by adequate surgery, it is curable in approximately three-fourths of the cases.

Two illustrations. EDSEL S. REED, M.D.
University of Louisville

Urinary Tract Changes in Cervical Carcinoma. William K. Diehl and J. Mason Hundley, Jr. Surg., Gynec. & Obst. **87:** 705–715, December 1948.

The authors briefly review the studies of others relative to the urinary tract in cervical cancer, indicating that some investigators have thought that damage to the ureters is principally due to extension of the disease, while others have blamed radiation therapy. To acquire evidence on this problem, a study was made of 37 patients with the several grades of cervical cancer, special attention being paid to the urinary tract before and after treatment. Retrograde pyelography was performed on all of these patients prior to the initiation of treatment and was repeated one year after treatment. The authors' technic of radium and roentgen therapy is briefly outlined.

Of the 37 patients, 10 (27 per cent) had demonstrable urinary tract dilatation before treatment, and it was established that the likelihood of pre-treatment dilatation was much greater in association with advanced lesions and that such dilatation was definitely allied to parametrial extension. Progression of the urinary tract abnormalities during the one-year period following diagnosis was shown to be a sign of grave prognosis, since all of the patients in this category died of cancer. In no patients with carcinoma of Grades 1 and 2 did demonstrable urinary tract changes develop in the period between examinations. On the other hand, 4 of 10 women with Grade 3 carcinoma without urinary tract involvement prior to therapy showed changes at the end of the year, and 3 of these died of cancer. None of the urinary tract dilatations present originally were reported to have improved following radiation therapy.

Attempts were made to determine the geometrical relationship of the pelvic ureters to the region heavily irradiated and also to determine the amount of irradiation necessary to produce deleterious ureteral scarring which might lead to stricture. The authors showed that by efficient vaginal packing during the radium treatment it was possible to push the uterus up and away from the relatively stationary pelvic ureters to a degree providing to the ureters considerable safety from the intense radiation. They clearly state their belief that their method of irradiation therapy will not produce ureteral stricture.

Fifteen roentgenograms; 1 table.
PAUL W. EYLER, M.D.
University of Pennsylvania

Treatment of Cancer of the Penis at The Norwegian Radium Hospital. Rolf Bull Engelstad. Am. J. Roentgenol. **60:** 801–808, December 1948.

Seventy-two histologically verified cases of cancer of the penis treated at the Norwegian Radium Hospital are reviewed. Sixty-four cases had primary irradiation therapy, and in 37 this was the only treatment aside

from preliminary operative correction of phimosis. Radium therapy (a mold or teleradium) was used, greatly protracted or protracted-fractionated. Regional lymphatics were always irradiated (usually with teleradium) whether or not metastases were clinically demonstrated. Dissection of groins was done for persistent, newly formed, or recurrent lymph node metastases. For local recurrent or persistent tumor, a comparatively conservative amputation of the penis was done or, in a few cases, electrocoagulation was used.

In this series of cases the percentage with freedom from symptoms was: for Stage I, 88.9 per cent of 36 cases at three years and 88.9 per cent of 24 cases at five years; Stage II, 67.9 per cent of 19 cases at three years and 63.6 per cent of 14 cases at five years. No cases of Stage III showed freedom from symptoms. For the total series, i.e., all stages, the figures were 70.8 per cent at three years and 66.7 per cent (of 57 patients) at five years.

According to the author, the advantages of the above plan of therapy are: that it can be carried out without complications; that the radiosensitivity is "rather good"; that favorable three- and five-year results as good or better than results after more radical treatment are obtained, and functional results are better.

Seven tables. O. RAYMOND RUSSELL, M.D.
Indiana University

Intra-Cavitary Contact Roentgen Therapy of Malignant Tumors. D. den Hoed. Acta radiol. **30:** 470–478, Dec. 31, 1948.

At the Rotterdam Radiotherapeutic Institute, where hundreds of cases of lip and skin cancer have been successfully treated by contact radiation therapy, the author has treated a wide variety of cancers by intracavitary application of contact radiation. The number of cases in which the method has been used is still small, however, and in many cases the follow-up period is insufficient to warrant statistical classification.

Seventeen case reports with summaries of total dosage given in each case and the current results are set forth. In many cases, intra-cavitary treatment was supplemented by deep external radiation, but in all cases the major dosage was contributed by the intra-cavitary contact method. This form of therapy has been used in cases of tumors of the mouth, nose, pharynx, hypopharynx, larynx, bladder, rectum, vulva, vagina, and uterus. In the naturally accessible cavities and also in those made accessible at surgery, the radiation is applied with a narrow contact roentgen tube.

The author maintains a high degree of optimism for the future use of this form of therapy but, at the same time, issues a warning against indiscriminate use of the method. He prefers that it be utilized only when other methods seem to be less successful or too dangerous.

W. C. GALLO, M.D.
Indiana University

Effective Dosage Levels and Interstitial Radium Therapy. Benjamin Jolles. Am. J. Roentgenol. **60:** 745–749, December 1948.

The distribution rules and charts for the Paterson and Parker dosage system answer two points of interest to the radium therapist: (1) How much radium is required? (2) How it should be arranged in any particular case?

The author attempts to take into consideration the area and volume of the surrounding normal tissues which exert an influence on the behavior of the tumor and makes suggestions for their consideration as a supplement to the Paterson and Parker dosage system. These considerations are believed to be of value since it is the surrounding normal tissues through which nutritional fluids pass into the tumor and through which decomposition products leave the tumor area. In previous work, the author investigated the effect of field shape on skin tolerance and concluded that the factor controlling the effect of the field was $\sqrt[3]{p/a}$, where p is the perimeter of the field and a the area. In a similar way, he considers the area of the shell surrounding an irradiated block of tissue and concludes that this factor is equal to $\sqrt[3]{\text{shell/vol}}$.

A table of relative effective dosage levels for cylindrical implants of various diameters is presented, and two illustrative cases are shown.

Four roentgenograms; 1 drawing; 1 table.

P. B. LOCKHART, M.D.
Indiana University

Carcinoma of the Anal Canal. Statistics of the Fondation Curie, Paris. J. L. Roux-Berger and A. Ennuyer. Am. J. Roentgenol. **60:** 807–815, December 1948.

Carcinoma of the anal canal as a separate entity was found at the Curie Foundation in Paris to have an incidence of only 0.58 per cent. No important five-year results have been published for this condition, making it difficult to estimate the comparative values of radiation therapy and surgical treatment. A review of the literature disclosed 10 five-year surgical cures and 11 five-year cures with radiotherapy.

The authors report 18 five-year cures in a series of 51 cases given radiotherapy between 1921 and 1940, with a complete analysis of results according to the extent of the primary lesion, presence or absence of lymph node metastases, and the histopathologic character of the tumor. Diverse methods used included radium implantation alone or combined with surface radium application, radium implantation plus intracavitary radium, and radium implantation plus teleradium.

Complications following radiotherapy included radionecrosis in 8 of 51 cases treated. In 7 of the 18 five-year cures there was stenosis or stricture. One severe case of infectious gangrene of the perianal tissue occurred and proved fatal.

Since 1940, two modifications of treatment have been introduced, namely, supervoltage and low-voltage or contact therapy, the latter for small lesions, the former for larger lesions extending outside the anal canal.

The authors feel that, although the percentage of failure in radiotherapy is still large, a much higher five-year cure rate can be attained if cases are treated early, when the lesions are small. Among 11 early cases treated in their series, there were 7 cures.

Two tables. WILLIAM C. GALLO, M.D.
Indiana University

Radiation Therapy in Peptic Ulcer: An Analysis of Results. William E. Ricketts, Walter Lincoln Palmer, Joseph B. Kirsner, and Anna Hamann. Gastroenterology **11:** 789–806, December 1948.

This is an analysis of the results of roentgen irradiation of the fundus and corpus of the stomach in more

than 800 cases of peptic ulcer studied from 1936 to 1947. Four technics were used, with certain identical factors, i.e., h.v.l. 1.5 Cu, 200 kv.p., 1 mm. Cu plus 1 mm. Al filtration, 20 ma., 50 cm. focal skin distance; intensity of approximately 33 r in air per minute. The depth dose to the gastric fundus varied from 666 to 2,500 r given over a period of six to fourteen days. There was a direct correlation between the decrease of hydrochloric acid secreted by the stomach and the total amount of radiation delivered.

In 47 cases of gastric ulcer it was found that a depth dose of 1,600 to 2,500 r would reduce acid secretion 50 per cent or more in 92 to 100 per cent of cases. In nearly two-thirds of these patients a true achlorhydria appeared within less than one month and persisted from a few days to eight years. In 21 cases it persisted thirty or more days.

Patients with duodenal ulcer were slightly less responsive: in 74 to 83 per cent significant achlorhydria developed after a similar depth dose of 1,600 to 2,500 r. A small series of jejunal ulcers was also treated.

The effects of the radiation therapy on healing of the peptic ulcer are summarized for 334 cases for which satisfactory studies were available. Healing was obtained in 93.8 per cent of those receiving a depth dose of 1,600 to 2,500 r. Of those receiving 666 to 1,000 r, only 70 per cent showed healing. All patients also received dietary and antacid therapy.

Recurrences of gastric ulcer were observed in one-third of the patients followed for longer than one year. This incidence contrasts with 80 per cent recurrence noted in gastric ulcer treated medically without radiation. Recurrences also occurred in approximately one-third of the cases of duodenal ulcer, excluding those given minimal radiation.

In conclusion, the authors state that irradiation of the acid-secreting portions of the stomach is a valuable procedure in the treatment of peptic ulcer, the effect being proportional to the reduction in gastric secretion. Achlorhydria of three months duration invariably results in healing of the ulcer. Recurrence is preceded by reappearance of acid gastric secretion.

Two photographs; 23 tables.

C. R. PERRYMAN, M.D.
Baton Rouge, La.

Radiation Therapy in Peptic Ulcer: A Study of Selected Cases. William E. Ricketts, Walter Lincoln Palmer, Joseph B. Kirsner, and Anna Hamann. Gastroenterology **11:** 807–817, December 1948.

The purpose of this paper is to focus attention upon the occasional dramatic effect of radiation therapy upon the course of chronic peptic ulcer and to correlate this effect with alterations in gastric secretion. It supplements the study abstracted above.

Three cases of gastric ulcer are presented illustrating healing of resistant ulcers after roentgen irradiation and one month or more of antacid therapy. Out of a series of 50 patients there were 5 in whom complete healing did not occur after irradiation. Only minimal gastric secretory depression was obtained in 3 of these; in 2 the achlorhydria was of short duration.

Two cases of intractable duodenal ulcer are also presented. Both cases showed healing after one or two series of x-ray therapy. It is apparently easier, however, to depress gastric secretion in patients with gastric ulcer than in patients with duodenal or jejunal ulcer. This is understandable, since the fasting secretory rate is on the average four times as great in patients with duodenal ulcer as it is in those with gastric ulcer. A few cases of jejunal ulcer have been subjected to radiation therapy. The results are often surprisingly good, but the incidence of ultimate recurrence has been high.

The authors conclude that the effect of roentgen irradiation upon the course of peptic ulcers depends upon the degree and duration of secretory depression.

Twenty roentgenograms; 5 charts.

C. R. PERRYMAN, M.D.
Baton Rouge, La.

Treatment of Uterine Fibroids. Franklin L. Payne. S. Clin. North America **28:** 1445–1455, December 1948.

Uterine fibroids begin as benign foreign bodies in the uterine wall and their growth is dependent upon stimulation from the ovarian secretion. As long as this stimulus lasts, they continue to grow, but when the stimulus disappears they begin to undergo regression.

The author divides the management of uterine fibroids into four general methods (1) observation, (2) hormone therapy, (3) irradiation, and (4) surgical removal. Each plan of therapy has its own indications and limitations and there is no competition among them. Symptoms or signs may develop at any time which will place the patient in one of the other groups. Conservatism in the choice of a plan and in its execution are essential to the proper management of myomas of the uterus.

Three drawings of surgical technic.

EDSEL S. REED, M.D.
University of Louisville

Evaluation of the Use of Beta Rays in Ophthalmology. James I. Moore. South. M. J. **41:** 1092–1094, December 1948.

This paper is based upon results obtained in the Radium Clinic of the Wilmer Institute of Johns Hopkins Hospital, where a radon beta ray applicator is used to treat eye lesions.

Beta ray therapy is considered the treatment of choice in the following conditions: vernal conjunctivitis; vascularization of the cornea following corneal ulcers, chemical burns, trauma, or keratitis; tuberculous keratitis and senile keratoses.

The following conditions can be treated successfully with beta rays, but respond equally well to other types of treatment; pterygia, keloids of the lids, and scarring of conjunctival flaps following surgery.

Conditions which do not respond to beta ray treatment include corneal dystrophies, old scars without vascularization, pigmented moles of the lids, sebaceous cysts, chalazions, and xanthelasma.

JOHN DECARLO, JR., M.D.
Jefferson Medical College

Prevention of Deafness by the Elimination of Hypertrophied Lymphoid Tissue in the Nasopharynx by Radium Therapy. J. Coleman Seal. New York State J. Med. **48:** 2715–2717, Dec. 15, 1948.

Gratifying results are reported by the author in the use of the Crowe type radium (beta ray) applicator for lymphoid tissue in the nasopharynx in general and around the eustachian tube orifice in particular. Not only does it prevent further middle ear damage but many

children are relieved of frequent upper respiratory infections and sometimes of allergic conditions.

Five cases are reported in detail.

Three drawings.

ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Radium Irradiation of the Nasopharynx for Hypertrophy of Lymphoid Tissue. B. H. Minchew and B. E. Collins. J. M. A. Georgia **37:** 439–443, December 1948.

The authors believe irradiation to be definitely indicated in cases in which lymphoid tissue obstructs the eustachian tubes. Of 35 patients with impaired hearing treated with the monel metal radium applicator during the past year, only 18 had received the full course of therapy. The hearing in 66 per cent of these cases had improved more than 15 decibels.

Treatment of Certain Types of Deafness by Roentgen Ray Therapy. Nelson A. Youngs and Philip H. Woutat. Ann. Otol., Rhin., & Laryng. **57:** 984–991, December 1948.

From 1937 to 1945, 116 patients were treated with deep roentgen therapy for deafness caused by lymphoid tissue obstructing the orifice of the eustachian tube, lymphoid infiltration along the tube proper, or in the middle ear cavity. The bases for selection of patients for this type of treatment were as follows:

1. The deafness must have been present longer than three months.

2. Lymphoid tissue could be demonstrated obstructing the orifice of the eustachian tube as observed through a nasopharyngoscope.

3 Those cases in which no lymphoid tissue could be demonstrated obstructing the eustachian orifice were treated only if a history of repeated attacks of otitis media, a demonstrable fluid level in the tympanic cavity, or retraction of the tympanic membrane were present.

The results of treatment were determined by tuning-fork tests, ability to hear whispered voice, and periodic audiograms in all cases except a few in which the subjects were too young to give a satisfactory audiogram. In these the results were judged on the ability to hear the whispered voice.

Group I (Age 3–15 years): In this age group there were 63 cases. Thirty-seven, or 58 per cent, obtained normal hearing after treatment.

Group II (Age 15–62 years): In this group involvement is most apt to be in the middle ear or along the eustachian tubes rather than in the nasopharynx or around the tubal orifices; 43.3 per cent of the group had chronic otitis media as compared to 20.6 per cent of those under 15.

All patients were treated with the following technical factors: 200 kv.p., 0.5 mm. Cu plus 1.0 mm. Al filtration, 50 cm. target-skin distance, h.v.l. 0.9 mm. Cu. Each treatment consisted of a dose of 125 to 150 r (in air) to each of two ports, one on each side of the face. The ports measured from 4 × 5 cm. to 5 × 6 cm., and were directed to include the middle ear, eustachian tube, and the nasopharynx. Four such treatments were given at weekly intervals. In 9 of this group (12 per cent) normal hearing was restored.

The average diameter through the head at the level of the ports is 12 cm. in seven-year old children, and 14 cm. in adults. With the factors used and a 20-sq. cm. port, the dose at 6 cm. and 7 cm. depth is 53 per cent and 44 per cent respectively of the dose in air at the skin. The dose to the skin and other normal structures irradiated is not enough to cause damage.

An occasional patient will have slight swelling and discomfort in the parotid gland starting a few hours after treatment. This subsides in a few hours. Dryness of the mouth or pharynx has not been noted.

Four diagrams; 2 tables.

STEPHEN N. TAGER, M.D.
Urbana, Ill.

Favus: Occurrence in Georgia—Report of Ten Cases. William L. Dobes. Urol. & Cutan. Rev. **52:** 729–734, December 1948.

Ten cases of favus of the scalp, 9 in a single family, are recorded. Seven of the patients were treated by roentgen epilation—400 r of unfiltered rays being given per area exposed. The five-point Kienboeck-Adamson method was used with the following factors: 100 kv., 6 ma., 12-inch target distance. The scalp was shaved prior to epilation. All crusts were washed off daily and 5 per cent ammoniated mercury ointment was applied twice daily for one week and once daily thereafter until the hair began to grow. Epilation was almost complete in four weeks, and in all cases there was regrowth of hair. In one case a reinfection after fourteen months received a second epilation.

Four photographs; 1 genealogical chart.

RADIOACTIVE ISOTOPES

Isotopes for Medicine. Paul C. Aebersold. J. A. M. A. **138:** 1222–1225, Dec. 25, 1948. **The Medical Profession and Atomic Energy.** Lewis L. Strauss. Ibid., pp. 1225–1227. **Medical Program of the Atomic Energy Commission.** Shields Warren. Ibid., pp. 1227–1228.

In this symposium on atomic energy, Aebersold presents data for the first twenty-two months of distribution of pile-producing isotopes, on the number of shipments, number of using institutions, and the number of individual projects in which the isotopes are being employed. Strauss outlines some of the problems of atomic energy development in the United States. Warren discusses briefly external radiation and internal radiation, radioactive isotope therapy, and the medical program of the Atomic Energy Commission. This program is divided into 4 parts: (1) the maintenance of health of the workers of the commission and its contractors; (2) the protection of environmental health; (3) the development of basic research; (4) the exploration of values of the new sources of energy available themselves and the value that very short-lived radioactive isotopes may have in cancer research and cancer therapy.

Graves' Disease: Treatment with Radioiodine (I^{131}). Mayo H. Soley, Earl R. Miller, and Nadine Foreman. J. Clin. Endocrinol. **9:** 29–35, January 1949.

In 1948 (M. Clin. North America **32:** 3, 1948. Abst. in Radiology **52:** 154, 1949) Soley and Miller presented

the results of treatment with radioactive iodine in 33 cases of Graves' disease. Now 68 patients have been studied and sufficient data are available to permit reporting the results in 46 of these.

A patient is considered to have responded satisfactorily to treatment by I^{131} if within approximately four months the signs and symptoms of thyrotoxicosis have disappeared, the thyroid has returned to normal size, and the basal metabolic rate, level of serum protein-bound iodine, and other laboratory findings are within normal limits. Forty-two patients fell into this category: the other 4 failed to show a satisfactory response. The failures are felt to be due to a conservative approach to therapy in the early phases of the study.

Animal experimentation was carried out to determine the effects of massive doses of I^{131} upon tissues surrounding the thyroid, and the pathologic changes are described. It was found that upwards of forty times the maximum therapeutic dose (in microcuries per kilogram) used in man is required to produce serious damage in contiguous structures in mice and rabbits.

The uptake of radioiodine by the thyroid in untreated Graves' disease was found to average 61 per cent, and in the treated patient 16.6 per cent. There appears to be a paradoxical lack of correlation between clinical response to treatment and the number of microcuries per gram delivered to the thyroid.

I^{131} in doses of 2 millicuries or more causes clinical and laboratory changes that point to tissue destruction (thyroid). After therapy with I^{131}, exophthalmos progresses less than in patients treated by subtotal thyroidectomy and more than in patients treated with x-rays. In 2 patients in this series myxedema developed.

EFFECTS OF RADIATION

Effect of Roentgen Irradiation on the Gastric Mucosa. William E. Ricketts, Joseph B. Kirsner, Eleanor M. Humphreys, and Walter Lincoln Palmer. Gastroenterology **11:** 818–832, December 1948.

This report describes a gastroscopic study of a series of peptic ulcer patients who received roentgen irradiation over the fundus and body of the stomach. The total quantity of radiation varied from 1,100 to 2,500 r, given in divided doses.

The characteristic gastroscopic features following irradiation consisted of redness and edema of the mucosa, hemorrhage, and adherent exudate. These changes were noted in 75 of the 77 patients examined one week to three months after irradiation, at a time when the gastric acidity was significantly reduced. The severity of this "irradiation gastritis" is directly proportional to the decrease in gastric secretion. The inflammation usually is transitory and is followed by atrophy, which is consistently present in patients with anacidity of two or more years duration. The nodular cobblestone-like features of "hypertrophic gastritis" usually disappear following roentgen irradiation, the mucosa assuming a normal or mildly atrophic appearance.

Development of severe "irradiation gastritis" in this group of patients was not manifested clinically by symptoms.

Illustrative case reports are included.

Thirteen illustrations; 5 tables.

C. R. PERRYMAN, M.D.
Baton Rouge, La.

Prolonged Achlorhydria Following Diagnostic Gastrointestinal Roentgen Studies: Report of a Case. M. C. F. Lindert and Michael F. Koszalka. Gastroenterology **11:** 930–933, December 1948.

The authors briefly report the case of a 30-year-old veteran in whom an achlorhydria developed and persisted for three and one-half years following repeated gastro intestinal roentgen studies. A post-irradiation ulcer on the skin of the lower left posterior thorax had been present for four years. The authors conclude, on the basis of one case, that a prolonged achlorhydria can be produced by irradiation but at the expense of damage to the skin and internal organs.

Three tables.

C. R. PERRYMAN M. D.
Baton Rouge, La.

Effect of Nitrogen Mustard and X Irradiation on Blood Coagulation. Leon O. Jacobson, Edna K. Marks, Evelyn Gaston, J. Garrott Allen, and Matthew H. Block. J. Lab. & Clin. Med. **33:** 1566–1578, December 1948.

A prolonged clotting time was produced in rabbits by the intravenous administration of 3 or 4 mg. of nitrogen mustard per kilogram of body weight and the amount of protamine necessary to produce clotting in the heparin tolerance test was increased. The mechanism of this prolonged clotting time and altered heparin tolerance is probably identical with the mechanism previously described following x-radiation, that is, an increase in a circulating heparin or heparin-like substance. This is substantiated by the facts that the prothrombin time, fibrinogen, and calcium levels were normal in the animals with a prolonged clotting time; no fibrinolysin was demonstrated; and antiheparin substances, such as toluidine blue and protamine, were capable of reversing to normal values or significantly reducing the prolonged clotting time for varying intervals. A recent report by these same workers has shown that therapeutic doses of nitrogen mustard produce this same effect in the human being (Smith, Jacobson, *et al.*: Science **107:** 474, 1948).

Another significant point found in this work is that following intoxication with either nitrogen mustard or x-ray, the clotting time is almost always significantly increased prior to significant reduction of platelet values. Spontaneous recovery of both the clotting time and the low platelet count occurs at practically the same time. Reversal of the clotting time and heparin tolerance to the normal range by the intravenous administration of toluidine blue or protamine sulfate did not significantly alter the platelet level. It is felt, however, that these facts do not necessarily indicate that platelets have no part in the production of these findings.

It is suggested that this potentially serious clotting defect may be produced by other agents producing toxic effects directly or indirectly on the blood-forming tissue. This should be borne in mind, since a method of treatment, namely, antiheparin substances, is available which may make it possible to pass the critical period of potential hemorrhage until spontaneous recovery ensues.

Nine charts; 1 table.

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Influence of Temperature on the Response of Frogs to X Irradiation. Harvey M. Patt and Marguerite N. Swift. Am. J. Physiol. **155:** 388–393, December 1948.

In a previous note (Federation Proc. **7:** 90, 1948), the authors reported that low temperature treatment of frogs after total body exposure to x-rays greatly prolongs their survival. The present studies were undertaken to determine (1) whether this altered radiosensitivity is due to a decrease in the rate of development of radiation injury or actually reflects a more rapid and complete recovery from the effects of irradiation and (2) whether body temperature during exposure influences the radiation reaction. Since the overt response of the frog, a poikilotherm, to x-radiation appears to be rather similar in many respects to that observed in the mammal, it was thought that these studies might be of practical as well as theoretical interest.

The authors' conclusions are as follows: toxicity is not influenced by altering body temperature of frogs during and/or for the first twenty-four hours after total body x-irradiation with 1,000 r and 3,000 r. Survival is greatly enhanced, however, as long as the animals are kept in the cold (5°–6° C.) continuously after the exposure. This altered sensitivity is due apparently to a decrease in the rate of development of radiation damage (prolongation of the latent period) rather than to any appreciable recovery. When the animals are removed from the cold after periods of sixty to one hundred and thirty days, there is no change in absolute survival nor any clear difference in the time course of deaths from that observed in irradiated animals maintained at 23° C. These findings indicate that the metabolic level during exposure does not influence the over-all radiation reaction in frogs and that the primary process of radiation damage is not repaired in the metabolically depressed animal.

Four graphs; 1 table.

Incidence and Origin of Androgenetic Males in X-Rayed Habrobracon Eggs. Anna R. Whiting. Biol. Bull. **95:** 354–360, December 1948.

In x-rayed Habrobracon eggs few or no deaths of androgenetic embryos occurred in spite of their development in cytoplasm irradiated with doses from 6 to 15 times that lethal for the egg nucleus.

It is definitely established that androgenetic development occurs in Habrobracon after irradiation in metaphase I. The percentage of eggs developing into androgenetic adults is low (1.57 per cent); 54,000 r is the highest dose at which androgenetic males were obtained, although the lethal dose for the egg chromosomes in their stage is 2,400 r.

Cytologic study of 294 such eggs showed 3 to be undergoing androgenetic cleavage while 3 others were possibly preparing for it. A maximum of 6, or 2.04 per cent, does not differ markedly from androgenetic survivors. It must be concluded that androgenetic embryos at the doses used are as viable as embryos developing in untreated eggs.

Chromatin bridges which appear in the mitotic division II after treatment in the first mitotic metaphase distort and retard the pronucleus of the egg, sometimes to such a degree that the sperm pronucleus cleaves and develops into a normal fertile haploid male with paternal traits only. The failure of androgenetic males developing in these eggs can be explained on the almost complete absence of the chromatin bridges after treatment by x-rays in the first meiotic prophase.

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Negative Effect of Folic Acid on Irradiation Leukopenia in the Cat. William S. Adams and John H. Lawrence. Am. J. M. Sc. **216:** 656–660, December 1948.

The title of this article very effectively summarizes the content. In the cat, under controlled conditions, following administration of both oral and subcutaneous folic acid before, during, and after whole body irradiation, there was no demonstrable difference in either the amount or severity of the leukopenia, which regularly occurred. Control animals not receiving folic acid responded almost identically to those that were given folic acid, both prophylactically and therapeutically. It is carefully pointed out that it should not be construed that these results are comparable to figures in which partial or regional roentgen therapy is employed. It is not inconceivable that some of the leukemias which developed during the course of prolonged roentgen treatment, particularly of the thorax and abdomen, result from interference in the production, absorption, or utilization of substances such as folic acid by virtue of the effects of roentgen rays on the gastro-intestinal tract.

Five charts; 3 tables. EVERETT L. PIRKEY, M.D.
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